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A MANUAL

OF THE

PRACTICE OF MEDICINE,

PREPARED

ESPECIALLY FOR STUDENTS.

BY

A. A. STEVENS, A.M., M.D.,

PROFESSOR OF PATHOLOGY IN THE WOMAN'S MEDICAL COLLEGE OF PENNSYLVANIA; LECTURER ON TERMINOLOGY AND INSTRUCTOR IN PHYSICAL DIAGNOSIS IN THE UNIVERSITY OF PENNSYLVANIA; PHYSICIAN TO ST. AGNES'S HOSPITAL AND TO THE OUT-PATIENT DEPARTMENT OF THE EPISCOPAL HOSPITAL, ETC.

—"is an arch where through
Gleams that untravelled world whose margin fades
Forever and forever as we move."

FIFTH EDITION, REVISED AND ENLARGED.

ILLUSTRATED.

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PREFACE TO THE FIFTH EDITION.

This edition has been thoroughly revised, and contains many important modifications and considerable additions. The chapter on Diseases of the Pancreas, the introductory chapter on Diseases of the Blood and of the Ductless Glands, and the articles on Appendicitis, Angina Pectoris, Aphasia, Myxœdema, and Syringo-myelia, have been entirely rewritten. New articles treating of Acute Cholecystitis, Tuberculosis of the Kidney, Gastroptosis and Enteroptosis, and Chronic Cerebral Leptomeningitis have been introduced. The author ventures to hope that the work in its present form may be found equal to existing requirements, and that it may prove as acceptable to students of medicine as former editions.

314 S. Sixteenth St., Philada.,
September, 1898.

PREFACE TO THE FIRST EDITION.

POPE says, "Half our knowledge we must snatch, not take." If this be true of general knowledge, it is certainly true of the knowledge of medicine as it is taught in the schools of to-day. In view of this fact, there seems to be a real need for books which present their subjects in an assimilable form.

At the request of many students the author has written this book with the hope that it may serve as an outline of Practice of Medicine, which shall be enlarged upon by diligent attendance upon lectures and critical observation at the bedside.

In its preparation the writings of the following authors have been freely consulted: Strümpell, Osler, Fagge, Bristowe, Frerichs, Liebermeister, Vierordt, Eichhorst, Wood, Ross, Gowers, Sansom, Henry, Tyson, Pepper, Paul, Murrell, Starr, Hilton, Duhring, Stelwagon, Van Harlingen, Tilbury Fox, Hardaway, Seiler, Cohen, Browne, Jacobi, Bruce, Brunton, Charcot, Dujarden-Beaumetz, Pavy, Mitchell, and Trousseau.

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DISEASES

OF THE

DIGESTIVE SYSTEM.

THE TEETH AND GUMS.

DELAYED DENTITION, and the eruption of badly-formed teeth, often result from rickets or congenital syphilis.

Caries of the teeth results from many conditions; notably, an unnatural softness of the teeth, lack of cleanliness, dyspepsia, the use of certain drugs, and diabetes.

Hutchinson's teeth.—The lateral incisors of the upper jaw are pegged, and the central incisors of the same jaw have convex sides, and crescentic notches on their cutting edges. These peculiarities indicate hereditary syphilis, and are noted only in the permanent teeth.

A blue line on the gums near the insertion of the teeth usually indicates chronic lead poisoning. Copper and silver poisoning occasionally produce similar lines.

Spongy, bleeding gums are often associated with scurvy. Swelling of the gums with tenderness and salivation is indicative of mercurial poisoning (ptyalism).

THE TONGUE.

Fur on the tongue.—This consists for the most part of accumulated epithelial cells, particles of food, and microörganisms, and results from an elevation of temperature or from disturbed innervation.

A light, uniform coat is often noted in health, particularly in those who sleep with the mouth open. Other causal conditions are:—

(1) Febrile diseases.
(2) Dyspepsia.
(3) Catarrhal conditions of the nose and throat.

Circumscribed furring often indicates local disturbance, as a jagged tooth or tonsillitis.

Unilateral furring may result from disturbed innervation, as in conditions affecting the second and third branches of the fifth nerve. It has been noted in neuralgia of those branches, and in fractures of the skull involving the foramen rotundum.

The dry, brown, and fissured tongue is noted in low fevers, as typhoid fever, typhoid pneumonia, typhoid dysentery.

A red, beefy tongue is noted in certain febrile diseases, as typhoid fever and scarlet fever, and in diabetes.

The "strawberry tongue" is characterized by a white fur, through which project bright red and prominent papillæ. It is seen in the early stage of scarlet fever.

A gray-coated and flabby tongue, with an oval bare spot in the centre, which is red and glossy, is sometimes seen in children, and is indicative of gastro-intestinal catarrh, or "mucous disease." (Starr.)

Tremor of the Tongue.

Trembling of the tongue is noted in many conditions; it is peculiarly marked in low fevers (typhoid), in alcoholism, and in paretic dementia.

Scars on the Tongue.

Scars on the tongue often result from syphilitic lesions, or from the tooth wounds of epilepsy.

FETOR OF THE BREATH.

This is often due to local inflammation, as chronic rhinitis, tonsillitis, etc.; to the retention of decomposing food, to caries

of the teeth, to certain lung diseases, especially gangrene and bronchiectasis, to dyspepsia, and to the ingestion of certain foods or drugs.

THE APPETITE.

Boulimia, or inordinate appetite, is a common symptom in nervous dyspepsia, hysteria, diabetes, and in certain insanities, notably in paretic dementia. It may be due to intestinal parasites.

Anorexia, or loss of appetite, is a symptom common to many conditions.

Pica is a craving for unnatural articles of food, and is noted particularly in chlorosis, insanity, and pregnancy.

DYSPHAGIA.

Dysphagia, or difficult swallowing, may result from: (1) Local inflammations. (2) Stricture of the œsophagus, spasmodic or organic. (3) Paralysis, local, as in diphtheritic paralysis; or centric, as in bulbar disease.

VOMITING, OR EMESIS.

Etiology.—(1) Toxic, from ptomaines, drugs, uræmia, and the specific fevers. (2) Centric disease, as cerebral tumors and meningitis; this type is often unaccompanied with nausea, and does not relieve the associated headache. (3) Diseases of the stomach, as ulcer, cancer, dilatation, dyspepsia, etc. (4) Reflex, as from pregnancy, uterine or ovarian disease, irritation of the fauces, worms, biliary colic, etc. (5) Intestinal obstruction; this is often fecal. (6) Disturbed cerebral circulation, as in swinging and sea-sickness. (7) Certain nervous affections, as hysteria, migraine. (8) Periodic vomiting may be in itself a neurosis, or may be associated with the gastric crises of locomotor ataxia. (9) Œsophageal vomiting results from obstruction, and the vomit is alkaline in reaction.

THE VOMIT.

Watery, or mucous vomit, is noted in chronic gastritis, in certain forms of nervous dyspepsia, and after persistent emesis, as in cholera.

Bilious, or green vomit, is not diagnostic of any special condition; it may occur in any case where vomiting and straining are continued.

Bloody vomit (*Hæmatemesis*).—For causes, see page 50. When present in large amount the blood can usually be recognized by the unaided eye; small amounts may be detected by the microscope, spectroscope, or by chemical tests.

Test for blood.—Evaporate some of the filtered coffee-grounds vomit in a watch-glass, scrape off some of the dried material; add a trace of finely-pulverized salt; place the mixture on an object-glass, and cover. Allow one or two drops of glacial acetic acid to run under, and again evaporate; when dry allow one or two drops of distilled water to flow under to dissolve the crystals of salt. Under the microscope minute brown rhombic crystals of hæmatin appear.

Purulent vomit may result from the rupture of an abscess into the œsophagus or stomach, or from phlegmonous gastritis.

Fecal vomit (stercoraceous) is indicative of intestinal obstruction. It is recognized by its odor and appearance.

Profuse vomit.—The ejection of large quantities of frothy fermented material is highly significant of gastric dilatation.

Vomiting without nausea, distress, or other gastric phenomena, occurs in certain neuroses of the stomach, in hysteria, uræmia, and in brain disease, as tumor, or as a precursor of apoplexy.

EXAMINATION OF THE GASTRIC CONTENTS.

The test-meal recommended by Ewald consists of an ordinary dry roll and two-thirds of a pint of water or weak tea, without milk or sugar. One hour after the ingestion of this meal about 40 c.cm. of fluid should be obtained from the stomach by expression. When, however, lactic acid as a pathological element is sought for, it is necessary to prescribe a meal which contains no preformed lactic acid. The one recommended by Boas is now commonly employed; it is a flour-soup consisting of a tablespoonful of oatmeal to a litre of water, and flavored with a little salt. Before administering this meal the stomach should be thoroughly cleansed of any existing residue that might mar the result of the test.

Test for Free Acids.—Filter-paper soaked in a solution of Congo-red, and dried, turns blue in the presence of free acids. A saturated alcoholic solution of tropæolin 00 turns from a brownish yellow to a dark brown when brought in contact with fluids containing free acids.

Qualitative Tests for HCl.—Günzsburg's phloroglucin-vanillin test will react with 1 part of HCl in 15,000 parts of water. The solution consists of 2 parts of phloroglucin, 1 part of vanillin, and 30 parts of absolute alcohol. When a few drops of this solution are heated with an equal quantity of the filtrate contained in a porcelain dish, a beautiful red color appears at the margin of the fluid. Boas states that the test is still more delicate when 100 parts of 80 per cent. alcohol are substituted for the 30 parts of absolute alcohol.

Boas' resorcin-sugar test gives a similar reaction. The reagent consists of 5 parts of resorcin, 3 parts of sugar, and 100 parts of diluted alcohol.

Total Acidity.—This is determined by allowing a decinormal alkali solution (water 10 c.cm., hydrate of potassium 56 milligrammes) to flow from a burette, drop by drop, into a beaker containing 10 c.cm. of filtered gastric juice, to which have been added as an indicator two drops of an alcholic solution of

phenol-phthalein. The test is completed when the red color produced no longer disappears on shaking the solution. Ten c.cm. of normal gastric juice usually require from 4 to 6.5 c.cm. of the standard alkali solution.

Since 1 c.cm. of the alkali solution is equivalent to 0.00364 gramme of HCl, it follows that the percentage of the latter in a given specimen will equal the number of c.cm. of the alkali solution required multiplied by 10, and again by 0.00364.

Test for Lactic Acid.—Dilute solutions of neutral ferric chloride turn canary yellow in the presence of lactic acid. *Uffelmann's reagent* is made by mixing one or two drops of pure carbolic acid with a few drops of dilute solution of neutral ferric chloride, and adding sufficient water to turn the solution a beautiful amethyst-blue color. Unfortunately other substances, such as sugar, alcohol, acid phosphates, and tartaric acid, give a somewhat similar reaction. The test is made more reliable by exhausting a portion of the gastric filtrate with pure ether, evaporating the ethereal extract, and finally testing an aqueous solution of the residue.

Boas' test, though somewhat complicated, is far more reliable. The gastric contents secured after the ingestion of the flour-soup test-meal are filtered, and if the presence of free acids be indicated by Congo-red, an excess of barium carbonate is added. The filtrate is then evaporated to the consistence of syrup, and the CO_2 is driven off by boiling with a few drops of phosphoric acid. The mixture is then thoroughly exhausted with ether that is absolutely free from alcohol, the ethereal extract evaporated, and the residue dissolved in 45 c.cm. of water. The aqueous solution is poured into a flask, and treated with 5 c.cm. of sulphuric acid and a small quantity of manganese dioxide. A bent-glass tube is made to connect the flask with a mixture of equal parts of a deci-normal iodine solution and a deci-normal sodium hydrate solution. On heating the contents of the flask to the boiling-point, the alkaline iodine solution becomes smoky and the odor of iodoform is detected when lactic acid is present.

Test for Acetic Acid.—This acid may be detected by its odor. The production of a blood-red color on the addition of a neutral solution of ferric chloride to an aqueous solution of

the ethereal extract which has been neutralized with sodium carbonate also indicates the presence of acetic acid.

Test for Butyric Acid.—This acid strikes a brownish-yellow color with Uffelmann's reagent. Its odor is also characteristic.

Test for Peptones and Propeptones.—These substances are the products of albumin digestion, and may be detected by the *biuret test.* When potassium hydrate and dilute copper sulphate are added to a solution of peptone a deep purple-red color is struck. With propeptone the reaction is the same; with albumin, however, the color is bluish-violet. The amount of peptone may be roughly estimated by first precipitating the albumin and propeptone by saturating the filtrate with crystals of ammonium sulphate, and then noting the intensity of the color reaction with the biuret test.

Test for Rennet.—This may detected by adding to 10 c.cm. of boiled milk having a neutral reaction an equal quantity of neutralized filtrate. When the mixture is treated in a water bath to a temperature of 30° to 40° C., a cake of casein forms in from 15 to 20 minutes.

Test for Pepsin.—Ewald recommends the following method of determining in a given specimen whether the pepsin or hydrochloric acid is present in too great or too small amount: An equal quantity of the filtrate is placed in four small test-tubes, and a disk of coagulated white of egg put into each. To the first nothing else is added; to the second 2 drops of hydrochloric acid is added for each 6 c.cm. of stomach contents; to the third from 0.2 to 0.5 gramme of pepsin is added; and to the fourth both the hydrochloric acid and pepsin are added. The test-tubes are then placed in an incubator at about 100° F. The rapidity with which the albumin is liquefied in the different tubes will indicate whether digestion would have occurred without having added anything, or whether acid or pepsin or both were necessary.

Test for Carbohydrates.—In health the digestion of starch is practically completed within an hour; after that time dextrins, maltose, and dextrose should be found instead of starch. If the last substance remain undigested it may be detected by the blue color which it strikes with Lugol's solu-

tion. With erythrodextrin the iodine solution gives a purple color, but with maltose and dextrose there is no reaction.

The Absorptive Power of the Stomach.—This is determined by the time required for free iodine to appear in the saliva after the ingestion of potassium iodide. The saliva is received on filter-paper impregnated with starch, a drop or two of fuming nitric acid is then added, and the appearance of a blue color proclaims the presence of iodine. Normally the saliva should yield the reaction for iodine in from ten to fifteen minutes after the ingestion of a capsule containing 0.1 gramme of potassium iodide. Care must be taken that none of the drug adheres to the outside of the capsule.

The Motor Power of the Stomach.—Ewald has suggested the use of salol, which escapes from the stomach into the intestine, where it is broken up into salicylic acid and phenol. Normally salicyluric acid appears in the urine in from forty to seventy-five minutes after the ingestion of one gramme of salol. Filter-paper moistened with urine containing salicyluric acid assumes a violet color when treated with a 10 per cent. ferric chloride solution.

ACIDITY OF THE GASTRIC CONTENTS.

Normal acidity is due to hydrochloric acid, but other acids are frequently formed during the digestive process, such as lactic, butyric, and acetic acids. The quantity of hydrochloric acid in normal gastric juice varies from 0.14 to 0.24 per cent., more acid being secreted after a heavy meal than after a light one.

Hyperacidity.—This condition is noted in chlorosis, in gastric ulcer, and in certain forms of nervous dyspepsia.

Subacidity or inacidity occurs: (1) In certain nervous affections, as in some forms of nervous dyspepsia, hysteria, and neurasthenia. (2) In extreme anæmia. (3) In gastric catarrh. (4) In gastric cancer. (5) In acute febrile diseases. (6) Often in passive congestion of the stomach, as from chronic heart and liver disease.

RUMINATION, OR MERYCISMUS.

Rumination is a condition, rarely observed in man, in which the food is regurgitated from the stomach and subjected to a second mastication. It is the result of a neurosis, and is generally found in association with hysteria, epilepsy, neurasthenia, or idiocy. It is sometimes hereditary, or acquired by imitation.

HICCOUGH.

Hiccough, or singultus, results from a clonic spasm of the diaphragm, and is often noted as a temporary condition after eating or drinking. Persistent hiccough is sometimes present in extreme exhaustion following acute or chronic diseases. It may also result from irritation of the phrenic nerve, as from the pressure of a thoracic aneurism. It may be reflex from stomachic, hepatic, intestinal, or peritoneal disease. It may be due to hysteria.

ABDOMINAL PAIN AND TENDERNESS.

Diffuse abdominal tenderness is noted in peritonitis, in hysteria, and in rheumatism of the abdominal muscles.

Persistent abdominal pain results from the various visceral diseases, chronic peritonitis, abdominal aneurism, and disease of the spinal vertebræ.

Colic is a painful spasm of a mucous canal. The common varieties are—biliary, intestinal, renal, uterine, and pancreatic.

Painful defecation results from constipation, anal fissure, dysentery, piles, ulceration, stricture, prolapse of the rectum, and inflammatory conditions of neighboring organs, as the uterus or prostate gland.

THE STOOLS.

Blood in the Stools (*Entrorrhagia or Melæna*).

The blood is nearly normal in appearance after profuse hemorrhages, or when it has been quickly discharged, as in piles and fissure. Retained blood imparts a black or tarry appearance to the stools.

Melæna results from: (1) Traumatism. (2) Acute inflammation of the bowels, as in enteritis and dysentery. (3) Obstructed circulation, as in chronic heart and liver disease. (4) Vicarious menstruation. (5) Blood dyscrasia, as in scurvy, purpura, infectious fevers, etc. (6) Rupture of an aneurism. (7) Ulcers in the intestines, as simple duodenal ulcer, typhoid, dysenteric, tubercular, or malignant ulcers. (8) Intussusception. (9) The passage of blood from the stomach in hæmatemesis. (10) Piles, fissure, fistula.

Watery, or serous stools are noted in choleraic diseases, in nervous diarrhœa, in the colliquative diarrhœa which terminates wasting diseases, in severe enteritis, and in corrosive poisoning, as by arsenic, antimony.

Green stools may result from an excessive amount of bile. They are also common in the diarrhœas of young children, and in these cases the green color may be due to bacterial growth. (Hayem.)

Black stools may follow intestinal hemorrhage, and the use of certain drugs, as charcoal, bismuth, iron, tannin, etc.

Red stools usually indicate blood, but they may be tinged red after the administration of hæmatoxylin (logwood).

Mucous stools are noted in intestinal catarrh, particularly when the lower bowel is affected, as in entero-colitis and dysentery.

Fatty stools result from the ingestion of large quantities of fats, from the absence of bile, and from chronic pancreatic diseases.

Purulent stools result from fistula in ano, dysenteric, syphilitic, or malignant ulceration, or the rupture of abscesses into the bowel, as prostatic and pelvic abscesses.

Lienteric stools. Stools which contain much undigested food are noted in inflammatory conditions of the stomach and upper bowel.

ABDOMINAL DISTENTION.

Causes.—(1) Enlargement of the various organs from tumors or other causes. Recognized by the history, irregular enlargement, and special symptoms referable to the organ affected. (2) Ascites. Recognized by movable dulness with superincumbent tympany, and fluctuation. (3) Tympanites. Recognized by universal tympany on percussion. (4) Pregnancy. Recognized by suppression of menses, morning emesis, pigmentation of mammary areola, softening of the cervix, intermittent uterine contractions, etc. (5) Distention of the bladder. Recognized by the history, location of dulness, and results of catheterization.

STOMATITIS.

Definition.—Inflammation of the mouth.

Etiology.—(1) Mechanical, chemical, thermal, or parasitic irritation. (2) Mercurial poisoning. (3) Cachectic states, as in phthisis, cancer, and diabetes. (4) It is most commonly seen in young children in association with gastro-intestinal disturbances, brought about by artificial feeding, warm weather, and bad hygienic surroundings.

Varieties.—(1) Catarrhal. (2) Aphthous. (3) Ulcerative. (4) Parasitic (thrush). (5) Gangrenous. (6) Mercurial.

General Symptoms.—Heat and pain in the mouth, increased flow of saliva, fetor of the breath, restlessness, languor, disinclination to nurse, and perhaps some fever.

Catarrhal Stomatitis (*Simple stomatitis*).

Symptoms.—General symptoms of stomatitis, and, on inspection, a diffuse red swelling of the mucous membrane.

Treatment.—Good hygienic conditions. Keep the mouth

clean. Employ a weak solution of silver nitrate, boric acid, or chlorate of potassium as a wash.

Aphthous Stomatitis (*Follicular stomatitis, Vesicular stomatitis*).

Symptoms.—General symptoms of stomatitis, and, on inspection, numerous small, round vesicles on the cheeks, lips, and tongue; these vesicles soon break, and leave little, shallow ulcers with a red areola.

Prognosis.—Good.

Treatment.—Sterilize the milk. Nurse at regular intervals. Wash the mouth with a clean linen cloth. Correct any gastric disturbance. Use locally:—

℞ Acid. boric., gr. x–xx;
Glycerini, f℥ss;
Aquæ, q. s. ad f℥ij.—M.

Chlorate of potassium (gr. xx–xxx) may be substituted for the boric acid.

Ulcerative Stomatitis.—This is thought by some to be an infectious disease, because it often occurs in epidemics, and attacks both children and adults when congregated and subjected to bad hygienic conditions.

Symptoms.—General symptoms of stomatitis.

Inspection.—The gums of the lower jaw are chiefly affected. They are swollen, red, and spongy. Linear ulcers, with gray, sloughing bases soon form, and may extend to the cheek. The glands under the jaw are swollen. In severe cases loosening of the teeth and necrosis of the bone may follow.

Prognosis.—Guardedly favorable.

Treatment.—Correct the hygiene. Tonic doses of quinine by the stomach or rectum are indicated. Touch the ulcers with nitrate of silver, and use as a mouth-wash a solution of chlorate of potassium or peroxide of hydrogen.

Parasitic (*Thrush, Muguet*).

Exciting Cause.—Saccharomyces albicans.

Symptoms.—General symptoms of stomatitis, and, on inspection, numerous milk-white elevations which, on removal, leave a raw surface. The disease may extend to the pharynx, œsophagus, and larynx. Microscopic examination reveals the fungus.

Prognosis.—Good.

Treatment.—Correct the hygiene. Treat any gastric disturbance. Tonics are often indicated. Locally, borax is of value, and may be used in the following mixture:—

℞ Sodii borat., ʒj;
Glycerini, fʒij;
Aquæ, fʒvj.—M.

Sig.—Apply several times daily by means of a camel's-hair brush.

Gangrenous Stomatitis (*Cancrum oris, Noma*).—This form is usually seen in debilitated children between the ages of two and six years, and usually follows one of the specific fevers, especially measles and whooping-cough.

Symptoms. — The general symptoms of stomatitis are marked.

Inspection.—The cheek is the part affected. Externally, it is swollen, hard, red, and glazed; internally, there is noted an irregular, sloughing ulcer.

Complications.—Perforation, septicæmia, lobular pneumonia from aspirated sloughs, and diarrhœa from the swallowing of fetid material.

Prognosis.—Grave. Death is common from exhaustion or complications. Recovery is often attended with deformity.

Treatment.—Good hygiene, alcoholic stimulants, nutritious food, tonics like iron and quinine.

Locally.—Evert the cheek and apply the actual cautery, or pack the surrounding parts with oiled lint, apply to the ulcer strong nitric acid, and subsequently neutralize with bicarbonate of sodium. As a mouth-wash, peroxide of hydrogen is of distinct value.

Mercurial Stomatitis (*Ptyalism*).—This form of stomatitis is seen in artisans who work in mercury, after the administration of very large doses of mercurials, and after the administration of small doses when there has been an unnatural susceptibility.

Symptoms. *Premonitory Symptoms.*—Tenderness of the gums, manifested by bringing the teeth forcibly together; redness of the gums near the insertion of the teeth, a metallic taste, and an increase of saliva.

Later Symptoms.—Profuse salivation, fetor of breath, redness, swelling, and tenderness of the gums. The tongue may be similarly affected and protrude from the mouth. In severe cases ulceration of the mucous membrane, loss of teeth, and necrosis of the jaw result.

TREATMENT.—Use astringent and antiseptic mouth-washes. Employ iodide of potassium in small doses to eliminate the mercury. Opium may be required at night to allay distress. Belladonna aids in arresting the secretion.

TONSILLITIS.

(Amygdalitis.)

ETIOLOGY.—Tonsillitis occurs at all ages, but it is particularly common in the young.

The rheumatic diathesis exerts a predisposing influence. Exposure to cold and wet usually excites it, and such exposure is very effective when the system is debilitated, or the throat is congested from improper use of the voice. Impure air, as the effluvium from foul drains or sewers, sometimes excites it.

VARIETIES.—(1) Simple, or catarrhal. (2) Follicular, or lacunar. (3) Phlegmonous (quinsy).

SYMPTOMS.—Pain in the throat increased by swallowing and talking; marked tenderness beneath the angles of the jaw; and fever with its associated phenomena; in severe forms the temperature is quite high, 104° or 105°.

In the *catarrhal* form the tonsils are uniformly swollen, red, and covered with tenacious mucus.

In the *follicular* form the tonsils are red and swollen, and present little yellow spots on their surfaces. These spots are found to be plugs of degenerated epithelium which are retained in the crypts on account of the swelling and occlusion of their outlets. These plugs are often expectorated during convalescence as offensive cheesy pellets.

In the *phlegmonous* form the tonsils are extremely swollen, often so much that they almost meet; the pain is intense and of a throbbing character. One gland soon becomes larger than the other, softens, fluctuates, and turns yellow from suppuration. Swallowing is almost impossible, the voice is lost, and breathing is difficult.

Diagnosis.—In children tonsillitis may resemble scarlet fever, especially when the former is associated with an accidental rash.

Scarlet Fever.—History of contagion, onset with vomiting, a punctated red rash, "strawberry" tongue, albuminuria, and pulse too rapid to be proportionate to the fever.

Diphtheria.—The follicular form resembles diphtheria, but in the latter there is a false membrane, not only on the tonsils but on surrounding parts, and its removal leaves behind a raw surface. The history of contagion, the rapid, weak pulse, the marked swelling of the submaxillary glands, albuminuria, and the Klebs-Löffler bacillus, detected by cultivation, will also indicate diphtheria.

Prognosis.—Favorable; even in grave cases rupture of the abscess occurs when death seems imminent. Suffocation from rupture during sleep, and death from ulceration of the carotid artery are extremely rare terminations.

Treatment.—Rest, light diet, and protection. In the beginning, salicylate of sodium (gr. xx thrice daily) may be given to shorten the attack. The ammoniated tincture of guaiacum (ʒij every two hours) is a very efficient remedy. The benzoate of sodium is also highly recommended:—

℞ Sodii benzoat., ʒj–ʒiv;
Glycerin.,
Elix. calisay., āā fʒj.—M.
Sig.—A teaspoonful every hour or two.

In some cases quinine (gr. v. thrice daily) with small doses of the tincture of aconite and the tincture of belladonna is an efficient remedy.

In severe cases opium is often required to relieve pain and to produce sleep.

Local Treatment.—Pellets of ice give much relief. The following remedies are efficient: Solutions of nitrate of silver, dry bicarbonate of sodium, guaiac lozenges (gr. ij), saturated ethereal solution of iodoform. Or:—

℞ Potass. chlor., gr. xx–xxx;
Tinct. ferri chlor.,
Glycerin., āā fʒss;
Aquæ, q. s. ad fʒij.—M.
Sig.—Apply several times daily with a camel's-hair brush.

When the glands are very much swollen scarification will lessen the pain and often shorten the attack. When fluctuation is detected the tonsil should be incised with a guarded bistoury.

External Applications.—An ice-bag, a poultice, or iodine.

HYPERTROPHY OF THE TONSILS.

ETIOLOGY.—Childhood, the rachitic and tubercular diatheses, and repeated attacks of acute tonsillitis are the predisposing causes. It may arise without obvious cause.

PATHOLOGY.—It may be a true hypertrophy, but in most instances either the glandular structure or the connective tissue predominates; and the firmness of the gland increases in proportion to the overgrowth of the latter. The follicles are often dilated, and filled with cheesy material which results from the accumulation of fatty-degenerated epithelium. Naso-pharyngeal catarrh and adenoid growths in the naso-pharynx are often associated conditions.

SYMPTOMS.—Difficult swallowing, mouth-breathing, snoring during sleep, a thick voice with a nasal twang to it, and malnutrition. Sufferers are very prone to acute attacks of catarrh of the nose and throat. In severe cases, from interference with breathing, the chest assumes the rachitic type—that is, flattened at the sides and base and prominent over the sternum.

PROGNOSIS.—Favorable under prolonged and careful treatment.

TREATMENT. *General Treatment.*—Build up the tone of the patient by frequent bathing with salt water, followed by friction, light gymnastics, deep breathing, and by the use of nutrient tonics such as cod-liver oil, hypophosphites, and iodide of iron.

Local Treatment.—A solution of nitrate of silver, or Lugol's solution (liquor iodi compositus), may be applied frequently to the tonsils; or dilute acetic acid (gtt. ij) or a dilute solution of iodine (gtt. ij) may be injected into the tonsils. When the glands are very large they should be removed by the tonsillotome, scissors, or galvano-cautery. Pharyngeal adenoids

should likewise be removed by the finger-nail or curette while the patient is under the influence of some general anæsthetic, or after the parts have been treated with cocaine.

PHARYNGITIS.

Acute Pharyngitis (*Acute "sore throat," Simple angina*).

Definition.—An acute catarrhal inflammation of the mucous membrane of the pharynx, soft palate, and uvula, and frequently associated with tonsillitis and laryngitis.

Etiology.—Exposure to cold and wet, especially when the system is debilitated or the throat is congested from improper use of the voice. It may be rheumatic in origin. It may be excited by local irritants, such as hot drinks or the inhalation of noxious gases.

Exposure to infectious fevers, like scarlatina and measles, may be followed by simple pharyngitis.

Symptoms.—Chilliness and slight fever with its associated phenomena; soreness in the throat, painful deglutition, a sensation of dryness or tickling, with a hacking cough; stiffness and tenderness of the muscles of the neck. Extension to the larynx may cause hoarseness; to the ear, through the Eustachian tube, deafness. Inspection reveals a red and swollen mucous membrane.

Varieties.—(1) *Simple;* recognized by the above symptoms. (2) *Rheumatic;* recognized by the history, intense pain, and stiffness of the muscles, without much change in the local appearance. (3) *Follicular;* the mucous membrane is red, swollen, and covered with whitish spots which represent retained secretion in the inflamed follicles. (4) *Infectious* pharyngitis is the form associated with the infectious fevers.

Prognosis.—Favorable.

Treatment.—Light diet and avoidance of exposure. Hot drinks, followed by Dover's powder (gr. x), and a saline purge will sometimes abort it.

Tincture of aconite (gtt. ij) with tincture of belladonna (gtt. v) every two hours is sometimes useful. In the rheumatic form the salicylate or benzoate of sodium is very efficient.

In simple angina Pepper recommends:—

℞ Potass. chlorat., ʒiss–ij;
Potass. bromid., ℥ss;
Ext. belladonnæ, gr. iij–v;
Syr. limonis, f℥j;
Syrupi, q. s. ad f℥iv.—M.
Sig.—Teaspoonful thrice daily.

Local Remedies.—A steam spray, pellets of ice, a gargle of chlorate of potassium (gr. x to f℥j), the application of a solution of nitrate of silver (gr. v to f℥j), or lozenges of cocaine, chloride of ammonium, or chlorate of potassium.

Chronic Pharyngitis.

Etiology.—Chronic "sore throat" usually results from repeated acute attacks, improper use of the voice, or the continuous action of irritants, like tobacco smoke.

Varieties.—(1) Hypertrophic. (2) Atrophic. (3) Ulcerative. (4) Phlegmonous.

Symptoms.—The voice is husky and its use is followed by distress; secretion is increased so that there is a constant desire to clear the throat; disagreeable sensations, as fulness, tickling, and the like, are frequently noted.

In the hypertrophic form (granular sore throat, clergyman's sore throat, chronic follicular pharyngitis) the mucous membrane is thick, swollen, traversed by dilated veins, and studded with numerous elevations which are composed of distended follicles and overgrown lymphatic tissue.

In the atrophic form (Pharyngitis Sicca), the mucous membrane is pale, smooth, glossy, and dry.

Ulcerative Pharyngitis.—Ulceration may be due to simple inflammation, syphilis, tuberculosis, cancer, and lupus.

Phlegmonous Pharyngitis (*Retropharyngeal abscess*).—Suppurative inflammation of the retropharyngeal connective tissue may occur as a sequel to one of the infectious fevers, or may be due to caries of the cervical vertebræ, or to the impaction of a foreign body.

It may be recognized by sore throat, weak voice, difficult deglutition, and the results of a digital examination.

Treatment.—Chronic pharyngitis does not result so much

from excessive use of the voice as from its improper use, and until this is corrected no treatment will be successful. Patients should be instructed to expel sounds by the aid of the diaphragm and abdominal muscles, instead of the muscles of the throat and larynx. The habit of hawking and scraping to clear the throat must be rigidly interdicted. The patient must guard against mouth-breathing. Sponging the neck night and morning, first with tepid, then with cold water, will render the throat less sensitive. The general health will require attention, and such tonics as iron, quinine, strychnine may be very useful.

Local treatment.—The naso-pharynx should be kept clean by frequent spraying or douching with some antiseptic solution like the following :—

℞ Sodii bicarb.,
Sodii biborat., āā gr. xx ;
Acid. carbolic., gtt. vj ;
Glycerin., f℥vj ;
Aquæ, q. s. ad f℥vj.—M. (Dobell.)

The nasal chambers should be inspected and any existing disease treated.

Astringent applications are often useful ; solutions of nitrate of silver, five or ten per cent., sulphate of zinc, or tannic acid, ten to twenty per cent., may be employed for this purpose. Lymphatic hypertrophies should be removed by the galvano-cautery.

Retropharyngeal abscesses will require evacuation and treatment directed to the cause.

Ulcerative pharyngitis will require appropriate constitutional treatment, and such local remedies as nitrate of silver, iodoform, nitric acid, etc.

STENOSIS OF THE ŒSOPHAGUS.

Varieties.—(1) Functional obstruction, due to spasm (œsophagismus). (2) Organic obstruction.

SPASM OF THE ŒSOPHAGUS.

Etiology.—Female sex; nervous temperament; hysteria; reflex irritation. It may occur as a symptom of hydrophobia, tetanus, and organic œsophageal obstruction.

Symptoms of Simple Œsophageal Spasm.—Paroxysmal dysphagia, often associated with a sense of constriction in the chest; little or no loss of flesh. An œsophageal bougie can be passed without much difficulty.

Diagnosis.—The age and sex of the patient, the paroxysmal character of the obstruction, the ability to pass a bougie, the absence of wasting, and the absence of any other cause, will serve to separate it from organic obstruction.

Prognosis.—Good for life, but indefinite as regards duration.

Treatment.—Search for some exciting cause and remove it when possible. The treatment is largely dietetic, hygienic, and moral. Tonics like iron, arsenic, and quinine are often indicated, and may be combined with such antispasmodics as valerian, asafœtida, or sumbul. The systematic passage of a bougie may be of great value. A mild electrical current may be applied through the bougie.

ORGANIC ŒSOPHAGEAL OBSTRUCTION.

Etiology.—(1) An external tumor pressing on the œsophagus. This is most commonly an aneurism. (2) A tumor growing from the œsophageal wall; generally a cancer. (3) A cicatrix, from ulceration. The ulcer may be due to syphilis or to some corrosive poison, as a strong acid or alkali. (4) A foreign body.

Symptoms.—A slowly increasing difficulty in deglutition, with the regurgitation of food. The œsophagus is often much dilated above the constriction, and the food may collect in the pouch thus formed, so that regurgitation may be delayed for several hours. The passage of a bougie meets with a permanent obstruction. There is much loss of flesh.

Diagnosis.—The history of syphilis or corrosive poisoning will suggest a cicatrix. Aneurismal obstruction can usually

be detected by physical examination. Aneurism should be excluded before a bougie is passed. The age, cachexia, pain, and involvement of other organs will indicate cancer.

Prognosis.—Depends on the cause. It is unfavorable in aneurism and cancer. In cicatricial contraction the obstruction may be overcome for an indefinite period.

Treatment.—Aneurism: Prolonged rest, restricted diet, and potassium iodide. Cicatricial contraction: Systematic dilatation with graduated bougies. Cancer: In the early stage, the cautious use of a bougie is advisable. In advanced cases the patient may be fed through a tube, and when this is no longer possible, life may be prolonged for a short time by rectal alimentation or by feeding through a gastric fistula.

ACUTE GASTRITIS.

(Acute Gastric Catarrh.)

Etiology.—(1) Ingestion of indigestible food, especially when followed by exposure to cold and wet. (2) Toxic substances in excess, as alcohol, strong acids, and alkalies. (3) It is an associated condition in certain infectious diseases, as yellow fever, measles, and scarlet fever.

Pathology.—The mucous membrane is red, swollen, and covered with thick mucus. It is sometimes the seat of ecchymoses.

Symptoms.—The symptoms vary much in degree. In severe cases there may be moderate fever (102°–103°) and its associated phenomena, with anorexia, coated tongue, intense pain in the epigastrium, which is tender to the touch, persistent vomiting, thirst, and considerable prostration. Jaundice may follow from the extension of the catarrh to the bile-ducts, and diarrhœa from its extension to the intestines.

Diagnosis.—It may resemble the onset of scarlet fever, but the history of contagion, the "strawberry tongue," sore throat, very rapid pulse, and eruption, characterize the latter.

Prognosis.—Usually favorable; it rarely lasts more than a few days.

Treatment.—Absolute rest. If the stomach has not been completely emptied, an emetic such as ipecac may be employed.

Locally, a mustard plaster or a turpentine stupe will aid in relieving the distress. In severe cases no food should be given by the mouth until the stomach becomes retentive. Thirst should be allayed with cracked ice. Later, milk with lime-water (a teaspoonful of each) may be given hourly, and this may be followed by light broths in similar quantities.

Persistent vomiting may be relieved by small doses of calomel (gr. $\frac{1}{12}$), bismuth (gr. v.-x.), carbolic acid (gtt. $\frac{1}{4}$-$\frac{1}{2}$), or wine of ipecac (gtt. 1).

℞ Hydrarg. chlor. mitis, gr. j ;
Bismuth. subnit., ʒj.—M.
Ft. in chart. No. xij.
Sig.—One every hour.

Or,

℞ Creosoti, gtt. iij ;
Bismuth. subnit., ʒj.—M.
Ft. in chart. No. xij.
Sig.—One every hour.

Or,

℞ Vin. ipecac.,
Tinct. nucis vom., āā fʒj.—M. (Pepper.)
Sig.—Two drops in water every two hours.

Severe pain and obstinate vomiting will often yield to opium, in the form of suppositories. Thus :—

℞ Pulv. opii, gr. vj ;
Ol. theobrom., q. s.—M.
Ft. in suppos. No. vj.
Sig.—One every three hours.

Toxic gastritis will require in addition appropriate antidotes.

DYSPEPSIA.

Definition.—The word dyspepsia means ill digestion, and is applied to a group of symptoms which accompanies every disease of the stomach ; when, however, the symptoms depend on nothing more than simple atony, hypersensitiveness, or chronic catarrh, the condition is spoken of as a distinct affection.

Corresponding to the latter view, three varieties have

been recognized, viz.: (1) Atonic. (2) Nervous, and (3) Catarrhal dyspepsia.

Etiology.—(1) Heredity. (2) All visceral diseases, as heart, liver, and kidney disease. (2) Overwork, mental or physical. (4) Gastric irritants, as tea, coffee, and alcohol in excess. (5) Dietetic errors, which include—insufficient mastication from bad teeth or hurried eating, too much food, insufficient food, coarse or improperly cooked food, excessive dilution of food with liquids, excess of condiments, and irregular eating.

Symptoms of Dyspepsia.—Coated tongue, perverted appetite, fulness and distress after eating, eructations, flatulence, "heart-burn," palpitation, headache, vertigo, disturbed sleep, and lassitude.

ATONIC DYSPEPSIA.

Characteristic Symptoms.—The tongue is pale, coated, flabby, and tooth-marked; the appetite is lost; there is a sense of fulness and distress over the stomach, some time after eating, without actual pain or tenderness. The bowels are constipated. There is much flatulence. The patient is pale, the muscles are soft, the pulse is weak, and there is great lassitude.

Prognosis.—Good.

Treatment.—The diet must be carefully regulated, and rich and heavy food rigidly interdicted. The hygienic surroundings must be so modified that the general condition of the patient will be improved. Tonics like iron, quinine, and strychnine are often indicated. Dilute mineral acids with pepsin will be required to assist the digestive process.

Purgatives should be avoided, and constipation relieved by diet, mineral waters, enemas, or suppositories.

NERVOUS DYSPEPSIA.

This type usually occurs in those of a distinctly nervous temperament, and excessive mental strain and dietetic errors are potent etiological factors. It is frequently associated with neurasthenia and hysteria.

Characteristic Symptoms.—The tongue is often clean. The appetite is very irregular—at one time it is lost; at another it is inordinate; at another it is perverted, the patient craving an unnatural diet. Vomiting is not common, but it may occur irrespective of the time of eating or of the character of the food. Pain is very variable, and may appear when the stomach is empty or full. In some cases *peristaltic unrest* is a prominent symptom. It is characterized by exaggerated peristaltic movements, perceptible to the patient, with borborygmi and gurgling. Gastric acidity is usually normal, but there may be subacidity or hyperacidity. The completion of the digestive act in the normal time after a test-meal is highly suggestive.

Other nervous phenomena are commonly present, such as headache, vertigo, disturbed sleep, hypochondriasis, neuralgia, palpitation, and perverted sensations.

Diagnosis.—The history, associated nervous phenomena, the time that the pain appears, the periods of complete relief, the absence of hemorrhage, cachexia, tumor, and local tenderness, are the chief diagnostic points.

Prognosis.—Good, when the cause can be removed and the patient thoroughly controlled.

Treatment.—The avoidance of excitement and excessive mental work must be enjoined. An extended voyage may effect a cure. In brain-workers the value of regular physical exercise and frequent bathing, followed by friction of the skin, cannot be overestimated. On the other hand, the anæmic and exhausted may require the "rest-cure." The patient's experience will assist in the regulation of the diet. Tonics like iron, arsenic, quinine, and strychnine are often indicated. Electricity applied to the stomach has given good results. Pepsin and mineral acids will be of service only in those cases in which examination reveals a lack of acid in the gastric juice. In such cases Dr. Pepper recommends:—

℞ Quininæ sulph., gr. xxxij;
Strychninæ sulph., gr. ss;
Acid. hydrochlor. dil., fʒij.
vel Acid. phosphor. dil., fʒiij;
Tr. cardamom. comp., fʒij;
Aquæ, q. s. ad fʒiv.—M. Filtra.
Sig.—Teaspoonful after meals.

CATARRHAL DYSPEPSIA.

(Chronic Gastritis, Chronic Gastric Catarrh.)

Catarrh of the stomach is often a primary condition resulting from the ordinary causes of dyspepsia, but its frequent dependence on disturbed circulation from heart, lung, and liver disease, or on some constitutional condition, such as anæmia, diabetes, tuberculosis, or Bright's disease, should never be forgotten.

Pathology.—In the early stages the mucous membrane is ashy-gray in color and covered with tenacious mucus. Ecchymoses are often noted. Microscopic examination reveals degeneration of the glandular epithelium and an overgrowth of the connective tissue. In advanced cases the walls may be thin from extreme atrophy of the glandular structure, but more often they are thick, wrinkled, and indurated from excessive overgrowth of connective tissue.

Characteristic Symptoms.—The tongue is irregularly coated, the tip often red, and the papillæ enlarged. The appetite is variable. After eating there is weight and distress, and often diffuse tenderness on palpation. There are frequent eructations of gas and sour liquid.

Nausea and vomiting are frequently present; the latter may occur in the morning on rising, and the ejected material be composed of the frothy mucus which has collected in the stomach during the night, or it may occur some time after eating, and be composed of partially-digested food mixed with acids of fermentation, such as lactic, butyric, and acetic acids. The normal acid, hydrochloric, is nearly always diminished in quantity. The bowels are constipated, and the urine is scanty and throws down a heavy deposit of urates or phosphates. The nervous phenomena common to all forms of dyspepsia are present.

Protracted cases, with atrophy of the gastric tubules, present the symptoms of pernicious anæmia.

Diagnosis. *Cancer*.—The age, hæmatemesis, cachexia, tumor, severe pain, persistent vomiting, the presence of lactic

acid after a Boas's test-meal, the short duration, and the involvement of other organs will suggest cancer.

Ulcer.—Hæmatemesis, sharp pain increased by eating, vomiting soon after eating, local tenderness, and abundance of hydrochloric acid will suggest ulcer.

Care must be taken to determine whether the catarrh is primary or secondary to visceral disease.

Prognosis.—When not dependent on organic disease of other viscera, the prognosis is good.

Treatment.—Good hygienic conditions. A regulated diet; in severe cases an absolute skimmed-milk diet, or partially-digested foods. Thick mucus and undigested food may be removed by the stomach-tube when its introduction is well borne. Pure or slightly alkaline water may be employed; but when there is much fermentation, one per cent. of salicylic acid may be added with advantage. Irrigation should be practised daily, or every other day, preferably before breakfast, and the tube should be kept in position until the escaping fluid is quite clear.

When lavage is not well borne, the patient may be directed to sip before breakfast a half pint of some hot alkaline water, such as Carlsbad. This is especially indicated when there is constipation.

Artificial Carlsbad salt:—

℞ Sodii sulph., ℥v;
Sodii bicarb., ℥ij;
Sodii chlorid., ℥j.—M. (Welch.)

Sig.—℥j in a half pint of water half hour before breakfast.

Dilute hydrochloric acid is nearly always indicated, and it may be combined advantageously with pepsin.

℞ Tinct. nucis vom., f℥ss;
Acid. hydrochlor. dil., f℥iij;
Pepsin., ℥iij;
Aquæ, q. s. ad. f℥iv.—M.

Sig.—A teaspoonful after meals.

The catarrhal process is often favorably influenced by subnitrate of bismuth, or nitrate of silver. When there is much fermentation and flatulence, salicylate of strontium (gr. v–x),

or subnitrate of bismuth with some antiferment may be employed.

℞ Salol, gr. xl;
Bismuth. subnitrat., ℥ss.—M.
Ft. in chart. No. xx.
Sig.—One powder half an hour before meals.

Instead of salol, creosote (gtt ½) may be added to each powder. Constipation should be relieved by diet, mineral waters, enemas, suppositories of glycerin or gluten, or by mild laxatives. Acid eructations and "heart-burn" may be relieved by digestants and dilute acids, taken immediately after meals; or by alkalies, with or without such antiferments as creosote, salol, or naphthol, taken one or two hours after meals.

GASTRALGIA.

(Gastrodynia, Neuralgia of the Stomach.)

Definition.—A painful paroxysmal affection of the stomach, unassociated with any organic lesion.

Etiology.—Nervous temperament, overwork, anæmia, and dietetic errors are the predisposing causes of simple gastralgia. A symptomatic variety is sometimes observed in gastric cancer and ulcer, in locomotor ataxia, and in nervous dyspepsia with hyperacidity.

Symptoms.—Paroxysms of severe pain in the epigastrium, usually radiating to the back, occurring when the stomach is empty, and relieved by pressure and the ingestion of food or warm stimulating drinks.

Diagnosis. *Gastric Ulcer.*—In this disease the pain is more continuous, is made worse by eating, and is often associated with local tenderness and hæmatemesis.

Cancer.—The age, history, continuous pain which is increased by eating, hæmatemesis, tumor, cachexia, anorexia, and absence of hydrochloric acid will separate cancer from gastralgia.

Angina Pectoris.—The radiation of the pain from the heart down the arm, fixation of the body, fear of impending death, and the associated symptoms of fatty heart, such as arcus

senilis, rigid arteries, and altered heart-sounds, will separate angina pectoris from gastralgia.

The lancinating pains of locomotor ataxia sometimes attack the stomach and produce what are termed *gastric crises.* These can be distinguished from simple gastralgia by the absence of the patellar reflex, by the Argyll-Robertson pupil, the loss of coördination, and by paroxysmal pains in other parts of the body.

Prognosis.—Favorable, but duration indefinite.

Treatment. *Attack.*—Hot fomentations should be applied locally, and Hoffmann's anodyne (℥ss), chloroform (gtt. x), dilute hydrocyanic acid (gtt. ij in hot water), or the following mixture may be given internally :—

℞ Spt. vin. gal.
Tinct. opii camph., āā f℥ss ;
Ol. caryoph., gtt. x.—M.
Sig.—A teaspoonful in hot water.

Or,

℞ Chloroformi,
Spt. ammoniæ aromat.,
Spt. vini gallici,
Tinct. cardamom. comp., āā ℥ss.—M.
Sig.—Teaspoonful every half hour.

In severe cases morphine will be required.

The Interval.—Correct the hygiene, regulate the diet, and enjoin rest. Travel may be extremely valuable. Neurasthenia may require the "rest-cure." Tonics are often indicated. When there is hyperacidity, salicylate of bismuth, carbonate of soda, or aromatic spirits of ammonia, after meals, may be very serviceable. Arsenic, strontium bromide (gr. x–xv), valerian, and dilute hydrocyanic acid are remedies of great value.

℞ Sodii arsenat., gr. ss ;
Ext. cannabis ind., gr. iij.—M. (DaCosta.)
Ft. in pil. No. xx.
Sig.—One, three times daily.

GASTRIC ULCER.

(Simple Ulcer, Perforating Ulcer.)

Definition.—An ulcer arising without obvious exciting cause, but which is probably due to the digestive action of highly acid gastric juice on a part of the stomach whose nutrition has been impaired by some local disturbance of the circulation.

Etiology.—Female sex, age (between the fifteenth and the fortieth year), overwork with poor food, and anæmia are the predisposing causes.

Pathology.—From some local disturbance of the circulation—injury, hemorrhage, thrombosis, embolism, or spasm of the vessels—the part is self-digested.

The ulcer is round or oval, usually situated at the pylorus, on the posterior wall, near the lesser curvature. It has a punched-out appearance, is conical in shape, with the apex towards the peritoneum, and is without an inflammatory areola. The floor of the ulcer is usually smooth, and may be formed by any one of the coats of the stomach. A series of ulcers is not uncommon, so that more than one may be detected.

Symptoms.—The general symptoms of dyspepsia, and the following characteristic symptoms: (1) *Pain.* This may be severe, appear soon after eating, radiate to the back, and be affected by position. (2) *Hemorrhage.* This appears in one-half of all cases; the bleeding may be profuse, and the blood bright red. (3) *Localized tenderness.* This is often detected by palpation two or three inches above the umbilicus. (4) *Vomiting.* This frequently occurs an hour or two after eating and at the height of the pain. (5) *Hyperacidity.* An increase of HCl is almost invariably noted after a test-meal, unless gastric catarrh be a prominent complication.

In some cases only the symptoms of dyspepsia are present, while in others all symptoms may be absent, and in the latter hemorrhage or perforation may be the first indication.

Events.—(1) Resolution. (2) Death from exhaustion, hemorrhage, perforation and peritonitis, or pyloric obstruction from cicatricial contraction.

Diagnosis. *Cancer.*—The age (after forty), history, down-

ward course, short duration, extreme cachexia, often out of proportion to gastric symptoms, tumor, absence of hydrochloric acid, and the presence of lactic acid.

Gastralgia.—The pain usually appears when the stomach is empty, and is relieved by food and pressure; no hemorrhage, no local tenderness; other nervous phenomena are commonly present.

Chronic Gastritis.—Hemorrhage rare, tenderness diffuse, pain less marked, vomiting less frequent and persistent, gastric acidity less than normal.

PROGNOSIS.—Guardedly favorable; such complications as hemorrhage or perforation may occur without warning, and relapses from new ulcers are not uncommon.

TREATMENT.—Absolute rest in bed and rectal feeding.

Later, and in less severe cases from the beginning, predigested milk, milk and lime-water, buttermilk, broths, soft-boiled eggs and preparations of corn-starch may be given by the mouth at regular and frequent intervals. This restricted diet should be continued for eight or ten weeks, and the return to solid food should be quite gradual. The more complete the rest the more rapid will be the cure. Lavage is contraindicated, but the stomach may be cleaned by the sipping of hot alkaline water in the morning before breakfast. Internally, subnitrate of bismuth and nitrate of silver are useful remedies.

℞ Argenti nitratis, gr. v;
Ext. opii, gr. iij.—M.
Ft. in pil. No. xx.
Sig.—One pill thrice daily half an hour before meals.

Or,

℞ Bismuth. subnitrat., ʒvj–ʒj;
Creosot., gtt. x;
Morphin. sulph., gr. i–ij.—M.
Ft. in chart. No. xx.
Sig.—One powder before meals.

Instead of morphine, cocaine (gr. $\frac{1}{8}$) may be added to each powder.

When there is much pain counter-irritation will be of service. Hemorrhage will require absolute rest; morphine (gr. $\frac{1}{8}$) and fluid extract of ergot hypodermically; an ice-bag to the

stomach, and pellets of ice and tannic acid (gr. v–x) by the mouth.

GASTRIC CANCER.

VARIETIES.—(1) Hard cancer (scirrhus). (2) Soft cancer (encephaloid). (3) Epithelioma. (4) Colloid cancer.

ETIOLOGY.—Male sex, age (after forty), heredity, and ulceration of the stomach are predisposing causes.

PATHOLOGY.—Cancer of the stomach is usually primary; other organs being involved secondarily. The columnar-celled epithelioma and the encephaloid are the most common forms. As the pylorus is the usual seat, gastric dilatation is a natural sequence.

SYMPTOMS.—The general symptoms of dyspepsia, with the following characteristic symptoms: Continued pain, often tenderness; vomiting of partially-digested food; absence of free hydrochloric acid in the gastric juice, and the presence of lactic acid after a flour-soup test-meal; hæmatemesis, the loss being usually slight, and the blood so altered by the gastric juice that it presents a "coffee-ground" appearance; presence of a tumor; loss of flesh and strength; extreme anæmia; involvement of the superficial lymph glands.

When the pylorus is involved, symptoms of gastric dilatation will be added. These are: Vomiting, after the lapse of several hours or days, of large quantities of fermented material; an increased area of gastric tympany on percussion, and a reversed peristaltic wave on inspection.

DIAGNOSIS.—The differential diagnosis of gastric cancer from ulcer, gastralgia, and chronic gastritis has already been discussed.

PROGNOSIS.—Absolutely fatal. The duration is from six months to two years.

TREATMENT. *Palliative.*—A liquid or semi-liquid diet. Rest. Hydrochloric acid and pepsin are often required to assist digestion. When the stomach is dilated lavage may give relief. Pain should be relieved by morphine. The other symptoms will require the treatment indicated in gastric catarrh. ~~At present, operative interference can scarcely be recommended.~~

PYLORIC OBSTRUCTION AND DILATATION OF THE STOMACH.

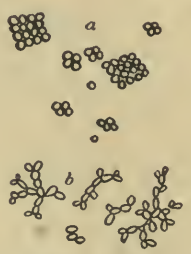
Etiology.—*The causes of pyloric obstruction:* (1) Pyloric tumors, usually malignant. (2) Tumors of adjacent viscera pressing on the pylorus or duodenum. (3) Cicatrix of an ulcer. (4) Fibroid thickening from chronic catarrh.

Pyloric obstruction increases the resistance offered to the expulsion of food, and in its efforts to overcome this, the stomach first becomes hypertrophied and then dilated.

Causes of Dilatation of the Stomach (*Gastrectasis*).—(1) Pyloric obstruction. (2) Relaxation of the walls from simple atony or catarrh. (3) Excessive ingestion of food or drink.

Symptoms.—The general symptoms of dyspepsia, with the following characteristic symptoms, most of which relate to the vomit: Vomiting occurs long after eating, sometimes several hours or days; the amount is often excessive, sometimes several quarts; it is sour and fermented, and on standing separates into a sediment of undigested food and a supernatant liquid, which is turbid and frothy; the ejected material is rich in torulæ and sarcinæ ventriculi. There is obstinate constipation.

Fig. 1.



a. Sarcina ventriculi. *b.* Torula cerevisiæ.

Physical Signs. *Inspection.*—Bulging over the epigastrium; in thin subjects the outline of the stomach may be visible. Sometimes a peristaltic wave is detected.

Palpation.—A splashing fremitus.

Percussion.—Increased area of gastric tympany. Artificial distention of the stomach with carbonic-acid gas, evolved by the administration of bicarbonate of soda and tartaric acid, is rarely necessary, and is sometimes harmful.

Auscultation.—Splashing sounds. These are often audible at some distance, and hence are a frequent source of annoyance to the patient.

Mensuration.—Normally an œsophageal sound may be inserted a distance of 60 ctm. from the teeth; in dilatation it may be inserted 65 or 70 ctm.

Prognosis.—Depends on the cause; it should always be guarded. It is more favorable in dilatation without obstruction. In cicatricial contraction operative interference has given fair results. In cancer the prognosis is absolutely unfavorable.

Treatment.—The diet should be light and nutritious, not bulky, and should be given in small amounts at frequent intervals. Lavage practised two or three times weekly is of great value. In cancer the treatment is palliative. In fibroid thickening and cicatricial constriction, dilatation of the pylorus (Loreta's operation) or the establishment of a gastro-duodenal fistula may be suggested. These operations have been fairly successful. In simple dilatation, treat the catarrh and apply massage and electricity; the latter may be applied to the interior of the stomach by means of a bipolar stomachal electrode. (Rockwell.) Tonics, especially strychnine, are often valuable adjuncts. An abdominal support often relieves some of the distressing symptoms.

GASTROPTOSIS AND ENTEROPTOSIS.

(Glenard's Disease.)

Definition.—A prolapse or downward displacement of the stomach and intestines.

Etiology.—The condition is most common in women. Tight-lacing, muscular strain, repeated pregnancies, rapid emaciation, and malnutrition are predisposing causes.

Pathology.—Gastroptosis and enteroptosis are frequently coexistent. Ptosis of the kidney, spleen, or liver may also be present. Both large and small intestines may be affected,

but the condition is more frequently observed in the colon, especially the transverse portion, which may be found elongated and tortuous, and occupying a position immediately above the symphysis pubis.

Symptoms.—When marked, ptosis of the viscera gives rise to nervous dyspepsia, flatulence, constipation, colicky pains, and the phenomena of neurasthenia. The position of the stomach and colon may be determined by inflation with air or gas.

Diagnosis.—After inflation the diagnosis between gastroptosis and *gastrectasis* can usually be made without difficulty.

Treatment.—The chief objects are to remove the cause and to improve nutrition. The application of massage and electricity to the abdominal walls may prove useful. A supporting bandage gives some relief.

HÆMATEMESIS.

(Gastrorrhagia.)

Etiology.—(1) Traumatism. (2) Acute gastritis. (3) Obstruction to the circulation, as in chronic heart, lung, and liver disease. (4) Vicarious menstruation. (5) Blood dyscrasia, as in scurvy, infectious fevers, grave anæmia, purpura, etc. (6) Rupture of an aneurism. (7) Gastric ulcer. (8) Gastric cancer. (9) Swallowing of blood from nose, mouth, or throat. (10) Hysteria.

Diagnosis. *Hæmatemesis.*—Blood is often clotted and mixed with food, is acid in reaction; the subsequent stools may be tarry, and the associated symptoms usually point to the stomach or adjacent organs.

Hæmoptysis.—Blood is red, frothy, and alkaline in reaction, the subsequent expectorations are streaked with blood, and physical signs usually indicate the cause.

Treatment.—Absolute rest; abstinence from food by the mouth; an ice-bag to the stomach. Pellets of ice may be sucked. Tannic acid (gr. v–x) by the mouth, and fluid extract of ergot (ʒss) with morphine (gr. $\frac{1}{8}$) hypodermically. If the hemorrhage has been profuse, use subcutaneous injections of weak saline solutions; give iron by the mouth, and advise the use of salty broths.

CONSTIPATION.

DEFINITION.—An unnatural retention of fecal matter.

ETIOLOGY.—(1) Many acute and chronic diseases which lessen peristalsis and secretion, as most chronic visceral diseases, all nervous diseases, anæmia, and the infectious fevers, except typhoid. (2) Sedentary habits. (3) Concentrated food. (4) Certain drugs, as lead and opium; it is an after-effect of strong purgatives. (5) Atony of the intestinal wall, common in the old and debilitated. (6) Stricture.

SYMPTOMS.—Infrequent stools, dyspepsia, fetid breath, headache, vertigo, lassitude, anæmia.

RESULTS.—In aggravated cases: dyspepsia, diarrhœa from irritation, fecal accumulation, hemorrhoids, fissure, fistula, prolapse of the rectum.

TREATMENT.—A regular time for defecation should be observed. Systematic exercise, abdominal massage, and electricity are valuable aids. Encourage the use of water, bran-bread, green vegetables, and stewed fruits. In mild cases a glass of water or an orange before breakfast will suffice. Enemata of water, or glycerine (℥j–℥iv), or suppositories of glycerine or of gluten may be required.

Mineral waters, like Friedrichshall or Hunyadi, often give relief.

In obstinate cases mild laxatives must be employed; cascara sagrada is one of the best. The dose of the extract is one to three grains; of the fluid extract, half to a fluid drachm.

Sometimes combinations are desirable.

℞ Aloin, gr. iv;
Styrchninæ, gr. ½;
Ext. belladonnæ,
Pulv. ipecac., āā gr.ij.—M.
Ft. in pil. No. xx.
Sig.—One or two as required.

Or,

℞ Pulv. rhei, gr. xl;
Pulv. aloes, gr. xx;
Ext. physostig., gr. iij;
Ol. caryophylli, gtt. iij.—M.
Ft. in pil. No. xx.
Sig.—One or two as required.

INTESTINAL COLIC.

(Enteralgia, Tormina.)

DEFINITION.—A painful spasmodic affection of the intestines.

ETIOLOGY.—It usually results from irritating food, flatulence, or fecal accumulation. It is sometimes of rheumatic or gouty origin. It may be reflex from disease of the ovaries, uterus, liver, spine, etc. It is also a symptom of lead-poisoning, intestinal inflammation, and intestinal obstruction. It may be a crisis of locomotor ataxia.

SYMPTOMS.—Paroxysms of severe pain of a twisting character, centering around the umbilicus, and relieved by pressure. The abdomen is usually distended. Severe attacks may lead to incipient collapse, indicated by cold sweats, pinched features, feeble pulse, and vomiting. The attack lasts from a few minutes to several hours, and usually ends by a discharge of flatus.

DIAGNOSIS. *Lead Colic.*—History, blue line on the gums, retracted abdominal walls, and lead in the urine.

Biliary Colic.—Pain radiating from the liver to the back and right shoulder, jaundice, and calculus in the stool.

Renal Colic.—Pain radiating down the ureter to penis and testicle, blood, mucus, pus, or calculi, in the urine.

Abdominal Aneurism.—Tumor, pulsation, *bruit.*

PROGNOSIS.—Favorable.

TREATMENT.—Apply hot applications to abdomen, and administer morphine (gr. $\frac{1}{8}$) with sulphate of atropine (gr. $\frac{1}{100}$) hypodermically. Subsequently employ a saline or mercurial purge. In the interval treat the causal condition.

Lead Colic.—Use magnesium sulphate as a cathartic, and potassium iodide (gr. v–x, thrice daily) to eliminate the lead.

DIARRHŒA.

DEFINITION.—A condition in which the stools are too frequent or too liquid. Like dyspepsia, it is a symptom of many pathological conditions.

ETIOLOGY.—(1) It results from inflammation of the in-

testines, as enteritis, entero-colitis, dysentery. (Inflammatory diarrhœa.) (2) It is a symptom of certain infectious diseases, as typhoid fever, cholera. (Symptomatic diarrhœa.) (3) It is produced by certain drugs, as laxatives and purgatives. (4) It may be an expression of cachexia occurring as a final symptom in cancer, diabetes, and chronic Bright's disease. (Colliquative diarrhœa.) (5) It may be a closing symptom in acute febrile diseases which end by crisis, as typhus fever, remittent fever. (Critical diarrhœa.) 6. It may result from nervous excitement or sensational disturbance. This is probably due to a vaso-motor paresis of the intestinal vessels (an intestinal "blush"), and the subsequent outpouring of serum. (Nervous diarrhœa.)

INTESTINAL CATARRH.

(Diarrhœa, Catarrhal Enteritis.)

ETIOLOGY.—Warm weather, childhood, and bad hygiene are general predisposing causes. It is usually excited by a sudden change in temperature, or by irritating products in the intestinal canal. Ptomaines produced by the decomposition of food are the most common excitants. It may be induced by corrosive poisons, as antimony, arsenic, mercury.

PATHOLOGY.—The mucous membrane, especially of the upper bowel, is injected, swollen, and covered with tenacious mucus. The solitary and agminated glands are enlarged, and are sometimes the seat of pinhead ulcerations.

In chronic enteritis the mucous membrane is often thickened from an overgrowth of connective tissue, but in some instances it is unusually thin from atrophy of the coats and destruction of the glands.

SYMPTOMS. *Acute Enteritis.*—Frequent stools, three to twelve or more a day, of a yellowish or greenish color, and containing undigested food; colicky pains, with rumbling noises (borborygmi); and slight fever with its attending phenomena.

Chronic Enteritis.—Frequent liquid stools which vary in color and character according to the seat of catarrh; much

undigested food (lientery) indicates involvement of the upper bowel; and much mucus, involvement of the lower bowel. The excessive drain leads to anæmia, emaciation, and weakness.

Membranous Enteritis.—This term has been applied to two conditions: (1) A true croupous enteritis, which is associated with the formation of a false membrane, and which is seen in cachectic states, in acute infectious diseases, and as a result of mineral poisoning. (2) Mucous colic, or mucous colitis, a chronic form of colitis, usually occurring in women of a marked nervous temperament, and characterized by paroxysms of severe pain, and the discharge of gray translucent casts which, however, are not membranous, but mucoid in character.

Diagnosis. *Dysentery.*—The marked prostration and tenesmus, and the small, mucous and bloody discharges will indicate dysentery.

Entero-colitis.—In this affection there is more fever, the prostration is greater, and the stools contain considerable mucus, and even blood.

Typhoid Fever.—The gradual onset, nose-bleed, splenic enlargement, characteristic temperature curve, and eruption will lead to the recognition of typhoid fever.

Prognosis.—Good, under favorable conditions.

Treatment.—In adults.—Rest. Liquid diet. When there is retention of irritating material, indicated by the history, sharp pain, abdominal distention, and small stools, administer a laxative, as calomel, or castor oil with laudanum.

℞ Hydrarg. chlor. mit., gr. ij;
Sodii bicarb., ʒj.—M.
Ft. in chart. No. xii.
Sig.—One every hour until five or six have been taken.

Or—

℞ Ol. ricini,
Syr. rhei aromat., āā fʒss;
Tinct. opii, gtt. x-xx.—M.
Repeat, if necessary.

When the bowel has been thoroughly emptied, opium, astringents, and intestinal antiseptics will be required. Thus:—

℞ Bismuth. subnit., ʒss;
Morphin. sulph., gr. j;
Creosoti, gtt. vj.—M.
Ft. in chart. No. xii.
Sig.—One every two hours.

Or—

℞ Bismuth. subnit.,
Cretæ præpar., āā ʒij;
Tinct. opii camph., fʒiss;
Tinct. kino, fʒij;
Pulv. acaciæ, q.s;
Aquæ cinnamomi, q.s. ad. fʒvj.—M.
Sig.—A tablespoonful every three hours.

Chronic Diarrhœa.—Liquid diet. Rest. Intestinal antiseptics (salicylate of bismuth, naphthalin, salol), and opium with mineral astringents.

Diarrhœa in Children.—Absolute cleanliness. Frequent bathing. A change of air, if possible. If the child is bottle-fed, the milk must be sterilized and given at regular intervals.

If the diarrhœa still persists, milk should be abandoned, and the child fed for a few days on egg albumin, beef juice, or beef peptonoids. A flannel binder should be applied to the abdomen. The bowels should be emptied with castor oil (ʒj) to which may be added a few drops of paregoric; or—

℞ Hydrarg. chlor. mit., gr. j;
Bismuth. salicylat., gr. xxxvj;
Pulv. zingiber., gr. xij.—M.
Ft. in chart. No. xii.
Sig.—One every hour.

After this has operated, astringents may be employed.

℞ Sodii salicylat., gr. xij;
Bismuth. subnit., gr. xxxvi;
Pulv. aromat., gr. vj.—M.
Ft. in chart. No. xii.
Sig.—One every two hours.

℞ Sodii bicarb., ʒss;
Syr. rhei aromat., fʒss;
Aq. menth. pip., fʒijss.—M. (Starr.)
Sig.—ʒj every two hours.

Or—

℞ Sodii salicylat., gr. xxiv;
Bismuth. subnit., ʒij;
Tinct. opii camph., fʒiij;
Mist. cretæ, fʒiss;
Aquæ cinnamomi, q.s. ad fʒiij.—M.

Sig.—One to two teaspoonfuls every two hours.

ACUTE ENTERO-COLITIS.

(Follicular Enteritis.)

Definition.—An inflammation involving mainly the ileum and colon, and affecting especially the lymphatic glands.

Etiology.—Warm weather, childhood, improper food, and bad hygiene are predisposing factors.

It may be a sequel of catarrhal enteritis or cholera infantum.

Pathology.—The mucous membrane is red, swollen, and œdematous. The solitary and agminated glands are swollen and often ulcerated.

Symptoms.—Frequent stools, at first yellow, later green, and mixed with curd, mucus, blood, and sometimes material resembling chopped spinach. The dejecta are neutral or acid in reaction. There is moderate fever 102°–103°, with its usual phenomena. The abdomen is distended, and tender along the colon. Vomiting is rarely persistent. The child grows pale, wastes, and assumes a senile appearance. Death may be preceded by coma and convulsions. (Spurious hydrocephalus.)

Diagnosis.—Reference has already been made to its separation from catarrhal enteritis.

Cholera infantum may be recognized by the abrupt onset, very high fever, incessant vomiting, serous purging, and early collapse.

Prognosis.—Grave, yet recoveries follow under favorable conditions.

Treatment.—Much the same as in catarrhal enteritis. Stimulants are frequently required. Weak stupes or spice poultices should be applied to the abdomen. Topical treatment should not be neglected. The bowel should be irrigated

once a day with a pint or more of cold water containing one per cent. of sodium benzoate or salicylic acid. The irrigation may be followed by the injection of an ounce of water containing nitrate of silver (gr. $\frac{1}{2}$–1) and perhaps laudanum (gtt. ij–iij).

CHOLERA INFANTUM.

Definition.—An acute disease of childhood, characterized by high fever, vomiting, purging, and collapse, and dependent upon an inflammation of the gastro-intestinal tract, and some disturbance of the sympathetic ganglia.

Etiology.—Hot weather, faulty feeding, dentition, and bad hygiene are predisposing factors.

Pathology.—The mucous membrane of the stomach and intestines is red, swollen, and œdematous; the glands are enlarged or ulcerated. The profuse serous discharges and rapid collapse must be due, in part, to some disturbance of the sympathetic nerves.

Symptoms.—The onset may be gradual or abrupt. Diarrhœa is usually the initial symptom; the stools are thin and serous, have a musty odor and an alkaline reaction. Vomiting soon develops, and the gastric irritability is so great that everything is rejected. Thirst is intense, the temperature is very high (105° to 108°); the pulse is rapid and feeble; the urine is scanty. Collapse follows, and is indicated by the pinched features, hollow eyes, sunken fontanelles, and cold surface. Even at this time a reaction may set in, but more commonly death results from exhaustion. The end may be characterized by the symptoms of spurious hydrocephalus—restlessness, convulsions, irregular pupils, and coma; and as these phenomena are unassociated with any cerebral lesion they are probably toxæmic.

Diagnosis. *Entero-colitis.*—Gradual onset, moderate fever, vomiting less marked, stools more mucous than serous and neutral or acid in reaction, pulse not so rapid, and no tendency to sudden collapse.

Prognosis.—Grave. Under conditions most propitious,

death may result in from one to three days; on the other hand, no aspect is too serious to admit of recovery. Entero-colitis is a common sequel.

TREATMENT.—If possible, the child should be removed to the country or seashore. It should be kept in the open air. Cleanliness is essential to success, and frequent bathing with cool water is desirable. A spice-plaster or a weak stupe should be applied to the abdomen.

The *nourishment* should consist of barley-water, beef-juice, wine-whey, chicken-broth, or frozen blocks of beef-tea; these should be given in small quantities at frequent intervals. Pellets of ice should be given to allay thirst. A few drops of brandy or of aromatic spirits of ammonia may be required at frequent intervals to combat prostration.

To arrest vomiting use calomel (gr. $\frac{1}{12}$), subnitrate of bismuth (gr. iij–v), or nitrate of silver.

℞ Argenti nitrat., gr. ss–j;
Syr. acaciæ, f℥j;
Aquæ, f℥ij.—M.
Sig.—A teaspoonful every two hours.

For the diarrhœa, laudanum (gtt. ij–iij) with starch-water (℥j) may be given every three or four hours by the rectum. Or the following may be given by the mouth:—

℞ Liquor. morph. sulph., f℥j;
Acid. sulphur. aromat., ♏ xxiv;
Elix. curacoæ, f℥ss;
Aquæ, q. s. ad. f℥iij.—M.
Sig.—One teaspoonful every two hours for a child six months old.

When vomiting and purging seem uncontrollable, morphine (gr. $\frac{1}{120}$ to $\frac{1}{100}$) hypodermically may be very useful.

Irrigation of the stomach and bowel with warm water has been highly recommended, and though heroic sometimes gives brilliant results. In collapse, use a hot bath to which a little mustard or red pepper has been added; then place the child in a horizontal position, cover with warm blankets, and administer stimulants freely.

DYSENTERY.

(Bloody Flux.)

DEFINITION.—An inflammatory disease of the colon, characterized by tenesmus, and the passage of small, mucous, and blood-streaked stools.

ETIOLOGY.—(1) Warm climates and warm weather; (2) bad hygience; (3) ingestion of irritating food; (4) exposure to cold and wet; (5) cachectic states (scurvy, gangrenous stomatitis, and Bright's disease) are predisposing factors, and alone may produce simple dysentery; but the tropical form (also occurs in cold climates) seems to be excited by an animal parasite, the amœba coli.

The disease frequently occurs in epidemic form.

VARIETIES.—(1) Acute catarrhal or sporadic dysentery. (2) Amœbic or tropical dysentery. (3) Malignant or diphtheritic dysentery. (4) Chronic dysentery.

PATHOLOGY. *Catarrhal Dysentery.*—Mucous membrane of the colon is red, swollen, œdematous, and in some cases ulcerated.

Fig. 2.

Amœba coli.

Amœbic Dysentery.—The mucous membrane is swollen from œdema and cellular infiltration. The latter causes superficial necrosis, and the formation of irregular ulcers which more or less undermine the surrounding mucosa. The amœbæ are found in the floor of the ulcers, and in the surrounding tissue. In some cases, false membrane and sloughs appear. Abscess of the liver is a common complication.

Diphtheritic Dysentery.—The mucous membrane is intensely swollen, and covered with a false membrane, which results from coagulation-necrosis. The separation of the membrane is followed by ulceration and sloughing.

Chronic Dysentery.—May be simple or amœbic. The coats are greatly thickened and ulcers are usually found. Cicatricial contractions sometimes follow.

Symptoms. *Acute Catarrhal Dysentery.*—Moderate fever and its associated phenomena, prostration, colic, abdominal tenderness, tenesmus (fulness in the rectum with a constant desire to defecate) with small, mucous, and bloody stools.

Amœbic Dysentery.—May begin as an acute or chronic disease. The symptoms are similar to catarrhal dysentery, but the disease is more protracted, and often marked by intermissions and exacerbations; the stools are more fluid and contain the amœba coli, and abscess of the liver is a more frequent complication than in other forms of dysentery.

Malignant or Diphtheritic Dysentery.—To the ordinary symptoms the following typhoid phenomena are added: Muttering delirium, stupor, subsultus, carphologia, and a brown, fissured tongue. The stools also contain false membrane and sloughs.

Chronic Dysentery.—Great loss of flesh and strength; extreme anæmia; the discharges contain considerable mucus and at times are bloody. Tenesmus and pain may be absent. The history of the initial symptoms will establish the diagnosis.

Diagnosis. *Diarrhœa.*—Absence of tenesmus and of mucoid and bloody stools.

Intussusception.—Late development of fever, stools more bloody than mucoid, the presence of a "sausage-like" tumor and persistent vomiting.

Prognosis.—In acute catarrhal dysentery the prognosis is good; recovery usually follows in from a few days to a week. In amœbic dysentery the prognosis should be guardedly favorable; relapses are common, and abscess of the liver is liable to occur. The duration in favorable cases is from six to eight weeks. Malignant dysentery is always a grave disease and often proves fatal.

Complications.—Peritonitis from extension or perforation, hepatic abscess, stricture, and paralysis from neuritis.

Hepatic abscess may be of amœbic or bacterial origin, although the former is the more common.

Treatment. *Acute Dysentery.*—Absolute rest and the enforced use of the bed-pan. Liquid diet. Apply externally

hot fomentations, mustard-poultices or leeches. A mild laxative is indicated in the beginning; sulphate of magnesium (ʒij), or castor-oil and laudanum might be selected, and either may be repeated until the effect is produced.

Internally, bismuth is a valuable remedy; salicylate of bismuth (gr. x) or subnitrate of bismuth with salol or creosote may be employed.

℞ Morphin. sulph., gr. j;
Bismuth. subnit., ʒij;
Creosoti, gtt. vj.—M.
Ft. in pulv. No. xii.
Sig.—One every hour or two.

Or,

℞ Salol, ʒj;
Bismuth. subnit.,
Sodii bicarb., āā gr. c.—M.
In twenty capsules. (DUJARDIN-BEAUMETZ.)
Sig.—One three or four times daily.

Musser recommends—

℞ Quininæ sulph., gr. xl;
Ext. opii, gr. v;
Mass. hydrarg., gr. x.—M.
Ft. in pil. No. xx.
Sig.—One or two every two or three hours.

In some cases, particularly in those associated with bilious symptoms, ipecacuanha, in large doses (gr. xx–xxx, repeated every three or four hours), is very serviceable. To prevent emesis, twenty drops of laudanum should be given half an hour before the administration of the ipecacuanha. Topical treatment should never be omitted. In mild cases opium suppositories will prove very beneficial; in severe cases enemata of thin starch-water with laudanum (gtt. xx–xxx) should be substituted for the suppositories. H. C. Wood highly recommends the use of ice suppositories, one every two to five minutes for half an hour, followed by suppositories of ergot and iodoform :—

℞ Ext. ergot., gr. lxxij;
Iodoform., ʒss;
Ol. theobrom., q. s.—M.
Ft. in suppos. No. vi.
Sig.—One every two hours until four or five have been taken.

Astringent injections of nitrate of silver or lead acetate should be reserved for subacute or chronic cases.

Injections of warm solutions of quinine ($\frac{1}{5000}$ to $\frac{1}{1000}$) have recently been employed in amœbic dysentery with advantage. (Osler.) Creolin (a drachm to the pint) has given good results in similar cases.

In malignant dysentery, quinine, alcohol, and turpentine are indicated.

Chronic Dysentery.—Rest; liquid diet; intestinal antiseptics (salicylate of bismuth), and copious injections of nitrate of silver in aqueous solution, as recommended by Wood. Begin with one or two pints (gr. xx to the pint), and inject through a tube pushed far up the bowel; later, increase to three or four pints (gr. xxx to the pint). The injections may be employed once or twice weekly.

CHOLERA MORBUS.

(English Cholera, Cholera Nostras.)

Definition.—An acute, sporadic disease, resembling Asiatic cholera, but not excited by the comma bacillus of Koch.

Etiology.—The summer season predisposes, and irritating food, as unripe fruit, and a sudden change of temperature are the usual exciting causes. A ptomaine or a special bacillus probably induces the disease.

Symptoms.—Intense cramps in the stomach, vomiting and purging of bilious material, moderate fever, and great prostration. In severe cases the discharges become serous, and symptoms of collapse develop.

Diagnosis. *Asiatic Cholera.*—No history of dietetic indiscretion; a direct etiological relation with another case; "rice-water" discharges; severe cramps in the legs, and presence of cholera spirilla.

Corrosive Poisons (as antimony).—History; the vomiting precedes purging; burning pain in œsophagus and rectum; and bloody mucous discharges.

Prognosis.—Favorable; death rarely occurs. Duration, twenty-four to forty-eight hours.

Treatment.—Hot applications to the abdomen. Morphine (gr. $\frac{1}{4}$) with atropine (gr. $\frac{1}{100}$), hypodermically, repeated if

necessary. When the pain is less severe opium may be given by the mouth or rectum. Ice is soothing and relieves the thirst. When vomiting is the most troublesome symptom the following will be beneficial :—

℞ Morph. sulph., gr. j ;
Creosoti, gtt. vj ;
Bismuth. subnit., ʒij.—M.
Ft. in chart. No. xii.
Sig.—One every hour.

Prostration will require stimulants, like aromatic spirits of ammonia or brandy.

In many cases the following mixture will be all that is required :—

℞ Tinct. opii camph., fʒss ;
Spt. ammon. aromat., fʒj ;
Magnesiæ, ʒj ;
Aq. menth. piperitæ, q. s. ad. fʒiv.—M.
(HARTSHORNE.)
Sig.—A teaspoonful every twenty minutes.

APPENDICITIS.

(Typhlitis, Perityphlitis.)

DEFINITION.—An inflammation of the appendix vermiformis.

PATHOLOGY.—There are three varieties : Catarrhal, ulcerative, and interstitial.

Catarrhal Appendicitis.—In mild cases the appearances are, no doubt, similar to those observed in catarrh elsewhere, but in severe cases the wall of the appendix is infiltrated with round-cells, and the mucous membrane is denuded of epithelium and presents a granular surface. This latter condition may eventuate in septic peritonitis, chronic appendicitis with relapses (*recurrent appendicitis*), or union of the granulating surfaces with complete obliteration (*appendicitis obliterans*).

Ulcerative Appendicitis.—In this type the wall of the appendix is the seat of a more or less localized ulcer. It may be associated with the presence of fecal concretion or a foreign body, or it may be the result of typhoid or tubercular infection.

Interstitial Appendicitis.—In this form the wall of the appendix is the seat of a necrosis, which is not infrequently gangrenous. It may be primary, infection taking place through the lymphatics, or secondary to the catarrhal or ulcerative form. It terminates in perforation, thereby exciting a most virulent type of peritonitis.

Appendicitis is always due to the action of pathogenic bacteria, the chief offenders being the bacillus coli communis, streptococcus pyogenes, staphylococcus pyogenes aureus, typhoid bacillus, and tubercle bacillus. Of these, the bacillus coli communis, a natural habitant of the bowel, is most commonly present. Under ordinary conditions it is harmless, but when the circulation of the appendix is interfered with from any cause or the coats of the tube are abraded, infection is liable to arise.

Etiology.—It is more common in males than in females. It is most frequent between the fifteenth and thirtieth years. Exposure, errors in diet, intestinal catarrh, traumatism, and the lodgement in the appendix of fecal concretions or foreign bodies predispose to the disease. It may follow some infection like typhoid fever, influenza, or tuberculosis. It may be induced by twisting of the appendix.

Symptoms.—(1) Sudden pain, often general at first, but later most marked in the right iliac region. (2) Circumscribed tenderness, most frequently detected over McBurney's point—a point midway on a line between the umbilicus and the anterior superior iliac spine. (3) Fever, ranging between 100° and 104° F. (4) Localized rigidity in the right iliac fossa, or the presence of a definite tumor. (5) Dorsal decubitus with the right thigh flexed. (6) Gastro-intestinal disturbances—anorexia, nausea, vomiting, constipation, or rarely diarrhœa.

Terminations.—Resolution, general peritonitis, and localized abscess. The location of the abscess depends on the position of the appendix. It may be found in either of the lower quadrants, or beneath the diaphragm (subphrenic abscess). The pus may be discharged through the abdominal walls, the bowel, bladder, or vagina, or it may escape into the tissues of the lumbar region or thigh. Appendicitis oc-

casionally excites hepatic abscess, the infection being carried through the portal vein.

Diagnosis. *Typhoid Fever.*—The gradual onset, characteristic temperature curve, epistaxis, mental hebetude, diarrhœa, splenic enlargement, and, later, the rash and Widal-reaction will indicate typhoid fever.

Renal Colic.—This may be recognized by the absence of fever and of local rigidity, and the presence of hæmaturia.

Acute Inflammation of the Gall-bladder.—Pain and tenderness in the right hypochondrium, a smooth, mobile tumor, and a history of biliary colic would suggest this condition.

Tubal Disease.—The history and results of pelvic examination will usually prevent an error in diagnosis.

Prognosis.—The prognosis depends on the type. The average mortality is about 14 per cent.

Treatment.—The patient should be confined to bed and placed upon a restricted milk diet. Warm or cold applications may be applied to the right iliac fossa. If there is much pain, morphine may be administered hypodermically. Troublesome constipation is best relieved by enemas.

Surgical intervention is required under the following circumstances: (1) At once in cases beginning suddenly with great severity. (2) In ordinary cases when no improvement is noted after the lapse of forty-eight hours. (3) At any time, should there be a sudden increase in the pain or a rapid diffusion of the tenderness. (4) Whenever a well-defined tumor can be detected in the right iliac region. (5) In cases recognized as tuberculous.

INTESTINAL OBSTRUCTION; ILEUS.

Etiology. *Acute Obstruction.*—(1) Congenital occlusion. (2) Intussusception (Invagination). (3) Strangulation, internal or external. (4) Twists (Volvulus) or Knots.

The following are conditions which produce *chronic obstruction,* though at times the symptoms develop acutely: (1) Stricture from a heated ulcer. (2) Unnatural accumulations, as fecal masses (Coprostasis), foreign bodies, gall-stones. (3) Tumors, within or without.

Symptoms. *Acute Obstruction*—(1) Sudden pain, at first paroxysmal, but later continuous. (2) Constipation. (3) Vomiting, persistent, and becoming fecal (stercoraceous). (4) Abdominal distention. (5) Collapse, indicated by pinched features, cold extremities, and feeble pulse.

Diagnosis. *Acute Generalized Peritonitis.*—The history, early appearance of fever and of diffuse tenderness, and the absence of persistent vomiting, especially of a stercoraceous character, will indicate peritonitis.

Chronic Obstruction.—These symptoms devolop slowly.

Congenital Occlusion.—The usual location is the anus or rectum. It is detected by direct examination.

Intussusception.—The slipping of a portion of intestine into another portion immediately below it. It is noted chiefly in children, and is more common in males. Its exciting cause is probably perverted peristalsis, whereby one part of the bowel is contracted while the adjacent part is dilated. In rare instances it has been induced by the traction of intestinal polypi. The usual seat is the ileo-cæcal region.

Multiple invaginations are frequently found post-mortem, which have resulted from the irregular peristalsis occurring just before death; they possess no inflammatory characteristics. In invaginations not cadaveric, the parts are injected, swollen, and covered with lymph.

Diagnosis.—The symptoms of obstruction, with the age; a "sausage-shaped" tumor in the line of the colon; the rare detection of the invaginated portion in the rectum; tenesmus; and bloody mucous stools are the diagnostic features.

Prognosis.—Death usually results from gangrene, peritonitis, or collapse. A favorable termination sometimes results from the escape of the incarcerated part, or by a sloughing off of the strangulated portion and adhesion of the serous surfaces.

Strangulation.—This often occurs in external hernia, when it can be recognized by an examination of the inguinal, femoral, and umbilical rings.

Internal Strangulation is due to the slipping of a coil of intestine through the diaphragm, foramen of Winslow, an abnormal opening in the omentum or mesentery, or a loop of inflammatory lymph.

DIAGNOSIS.—It might be suspected by the absence of other cause, by the sudden onset, or by a history of previous peritonitis.

Twist.—Occurs most commonly in middle-aged men. The usual seat is the sigmoid flexure. A relaxed and lengthened mesentery is a predisposing factor.

DIAGNOSIS.—Rarely possible.

Stricture.—Usually results from syphilitic, tuberculous, or dysenteric ulcers. The rectum is the most common seat.

DIAGNOSIS.—Based on the history, gradual onset, results of rectal examination, grooved or ribbon-like stools, bloody discharges, and visible peristalsis.

Unnatural Accumulations.—Fecal impaction is recognized by the gradual onset, mild obstructive symptoms, history of constipation, and a painless, irregular, doughy tumor in the line of the colon.

Gall-stones may obstruct the ileum; the history will aid in their recognition.

Tumors.—The most common tumor within the bowel is a cancer; it is usually located in the sigmoid flexure or rectum.

DIAGNOSIS.—Age, gradual onset, pain, bloody discharges, cachexia, and a tumor in the rectum are the characteristic features.

Tumors of adjacent viscera may compress the bowel. Their recognition will depend upon physical examination.

TREATMENT OF ACUTE OBSTRUCTION.—Food by the mouth should be withheld. Ice may be given to quench thirst. Nutritive enemata should be employed in the weak. Cathartics are contraindicated. Pain is best relieved by the administration of morphine hypodermically. Washing out the stomach three or four times daily is recommended for the persistent vomiting. Distention of the large bowel with gas or fluid should be practised in doubtful cases and in intussusception. It may be accomplished by placing the etherized patient in the knee-elbow position and administering warm water by means of a fountain syringe, elevated according to the patient's age from six to fifteen feet, the nozzle being carried well up into the bowel. Senn recommends inflation

with atmospheric air or hydrogen gas. After failure with these methods celiotomy should not be delayed; the earlier its performance the greater the chance of success.

In *fecal impaction* injections of warm water, oil, or an aqueous solution of ox-gall are efficient. Salines may be administered by the mouth. Electricity is sometimes useful. Rectal accumulations may be removed by the fingers or a suitable scoop.

Strictures require surgical interference.

ANIMAL PARASITIC AFFECTIONS.

Tape-worms.

VARIETIES. — Tænia solium. Tænia saginata. Bothriocephalus latus. Tænia echinococcus.

HISTORY.—The eggs of the tape-worm are ingested by an animal, and embryos, or proscolices, are liberated in the stomach; these migrate to other organs, where they are transformed into larvæ or scolices. The encysted larva, or scolex, is termed a cysticercus; the condition is known as "measles." The mature worm develops in man from the cysticercus contained in infected meat.

Tænia Solium (*Pork Tape-worm*).—Is derived from the hog, and is two or three yards in length. The head is the size of that of a pin, is provided with four pigmented cup-like suckers, surrounded by a double row of hooklets, and is attached to the body by a thread-like neck. The sexual orifice is in the centre of the broad surface of the segment.

Tænia Saginata (*Tænia Mediocanellata*).—Is derived from beef, and is five or six yards in length. The head is larger than that of the tænia solium, and has four large suckers, but no hooklets. The segments are fatter, and the uterine branches are finer and more numerous than in the tænia solium.

Bothriocephalus Latus.—Is found especially in Europe, and is derived from fish. The head has no hooklets, but two lateral grooves. The body is very long. The sexual orifice is on the narrow side of the segment.

SYMPTOMS.—Often absent. Frequently there are dyspeptic symptoms, colicky pains, loss of flesh, capricious appetite, and sometimes reflex nervous phenomena, such as vertigo, palpitation, "night-terrors," convulsions, itching in the nose, and choreic movements.

The DIAGNOSIS rests on the discovery of the eggs or segments in the stools.

TREATMENT.—A light diet for a day or two, and a saline purge prior to the administration of the anthelmintic. After an unsubstantial breakfast administer one of the following efficient remedies: Pumpkin seeds (two to three ounces); oleoresin of male fern (ʒj–ij), pelletierine, the alkaloid of pomegranate (gr. v); Kooso (ʒss).

℞ Oleoresin. aspidii, fʒj;
Pulv. acaciæ et sacchar., āā q. s.
Aquæ cinnamomi, q. s. ad fʒij.—M.
Sig.—One tablespoonful, repeated if required.

A purge should be given a few hours after the vermifuge. The treatment is successful only when the head is passed.

Nematodes.

Ascaris Lumbricoides (*Round Worms*).—Life history unknown. They are of a pale-pink color, and in form resemble earth-worms. They inhabit the small intestines, but occasionally migrate into other organs, viz., stomach, bile-ducts, and larynx. They are most commonly found in children.

SYMPTOMS.—Often absent. Sometimes there are dyspepsia, mucous stools, colicky pains, voracious appetite, anæmia, and reflex nervous phenomena, as "night-terrors," grinding of the teeth, pruritus of nose and anus, choreic movements, and convulsions.

TREATMENT—Santonin (gr. ¼–gr. iij); worm-seed oil (gtt. x in capsule or on sugar); fluid extract of spigelia (fʒj–fʒiij), are efficient remedies.

℞ Santonini, gr. vj;
Hydrarg. chlor. mit., gr. vj;
Sacchari., gr. xxiv;
M. et ft. chart. No. xij. (STARR.)
Sig.—One powder morning and evening.

Oxyuris Vermicularis (*Seat-worm, Pin-worm*).—This is a small worm, most commonly seen in children, and occupies the colon and rectum. It produces intense itching of the anus, which is worse at night. It may migrate into the vagina and excite pruritus or vaginitis, and lead to masturbation.

Treatment.—An injection of water, followed by the injection of two or three ounces of an infusion of quassia chips (℥ij–iij to the pint).

Anchylostomum Duodenale.—A small worm, not uncommon in the north of Europe and Egypt. It has been detected most frequently in miners and brickmakers, who are probably infected by drinking water containing the eggs of the parasite. The worm inhabits the small intestine.

Symptoms.—Dyspepsia and intense anæmia. The latter has been termed Egyptian chlorosis, and may be recognized by the detection of eggs in the stools.

Treatment.—Santonin, male fern, and thymol have been recommended.

Tricocephalus Dispar (*Whip-worm*).—A small worm, thick at one end and thread-like at the other. It occupies the colon and cæcum, and produces but little disturbance.

Filaria Sanguinis Hominis. — A small thread-like worm, most commonly seen in the tropics. The adult occupies the lymphatics, and the female brings forth a great number of embryos, which soon find their way into the blood-current. The embryos of the most important species of filaria (*Filaria Bancrofti*) are found in the blood only at night. The medium of infection is probably the mosquito, which carries the embryo from the blood to the water.

Symptoms. — Often absent. Chyluria, hæmaturia, and lymph-scrotum sometimes result from lymphatic obstruction.

Trichina Spiralis.—A small worm derived from the hog. Man is infected by eating insufficiently-cooked pork containing the encapsulated worm. The worm is set free in the stomach, where it develops and brings forth living embryos. These soon migrate into the muscles, where they in turn develop, coil themselves up, and become encapsulated. Trichinous capsules, impregnated with lime-salts, are visible to

the naked eye, and are sometimes detected accidentally at autopsies.

Symptoms of Trichinosis.—Sometimes absent. When large numbers have been ingested, *gastro-intestinal symptoms* develop in a few days. These are: Pain, nausea, vomiting, and serous diarrhœa.

Muscular Symptoms.—In from one to two weeks muscular symptoms develop. The muscles become swollen, firm, extremely tender and painful. Movement is inhibited, and dyspnœa results from the involvement of respiratory muscles. Œdema, especially of the face, is a prominent symptom. Profuse sweating is sometimes observed, and high fever is commonly present.

Blood.—Examination of the blood shows a marked increase in the eosinophiles.

Prognosis.—Depends on the number of worms ingested. The majority of patients recover.

Treatment.—Prevent by thoroughly cooking all pork products. In the first stage use purgatives. After migration employ opium, warm fomentations, and stimulants.

PERITONITIS.

Definition.—Inflammation of the peritoneum.

Varieties.—According to cause, it may be primary or secondary; according to extent, local or general; according to time, acute or chronic; and according to the exudate, sero-fibrinous, fibrinous, or purulent.

Etiology.—Acute peritonitis may be: (1) Idiopathic, arising from exposure to cold and wet (rare). (2) Traumatic. (3) Perforative, resulting from a perforating wound, or the rupture of a gastric, typhlitic, typhoid, or dysenteric ulcer, or a visceral abscess. (4) Secondary to inflammatory disease of adjacent viscera, as septic endometritis and typhoid fever. (5) Secondary to some general morbid process, as rheumatism, Bright's disease, scarlatina, tuberculosis, or variola.

Pathology.—In the first stage the membrane is red, sticky, and lustreless; later, a sero-fibrinous, fibrinous, or puru-

lent exudate is formed. In some cases the exudate is tinged with blood.

SYMPTOMS. *Acute General Peritonitis.*—Chill; moderate fever (102°–103°), with its associated phenomena; a rapid, wiry pulse; abdominal pain and tenderness so intense that abdominal respiration and body movements are inhibited; the patient lies on his back with his thighs flexed; the features are pinched; the vomiting is persistent; the bowels are usually constipated. Hiccough is a common and troublesome symptom.

Inspection reveals great abdominal distention.

Palpation elicits tenderness, and rarely a friction fremitus.

Percussion at first yields universal tympany; but later, dulness in the flanks from the gravitation of the exudate.

DIAGNOSIS. *Acute Enteritis.*—Pain and tenderness not so marked, absence of wiry pulse, and diarrhœa instead of constipation.

Intestinal Obstruction.—Unless associated with peritonitis, there is no fever, no wiry pulse, nor extreme tenderness; the vomiting becomes fecal.

Hysterical Abdomen.—This condition may resemble peritonitis in all particulars. The sex and personal history must be considered. Fever is not usually present, the pulse is not rapid and wiry; when the attention is distracted the pain may vanish.

PROGNOSIS.—Generally unfavorable. Death usually results in a few days from exhaustion. When the process is neither septic nor extensive recovery frequently follows.

TREATMENT.—Restrict the diet. Administer opium in full doses to check peristalsis and relieve pain. In severe cases the drug may be pushed until the respiration has been reduced to twelve per minute; apply leeches to the abdomen, and follow with light poultices. In some cases cold cloths are more grateful than warm applications. In non-perforating cases, salines, as Epsom or Rochelle salts (℥ij), may be given until bowels move freely. These salts, while not increasing peristalsis, attract serum from the turgid bloodvessels, and so relieve congestion. In perforating cases—and these are the most frequent—laparotomy offers the only hope of cure.

Chronic Peritonitis.

Etiology.—It is usually tuberculous; it may be cancerous; it may be syphilitic (occurring in young children); it rarely follows Bright's disease it rarely follows an acute attack; it occurs in chronic alcoholism.

Pathology.—The intestines are matted together by bands of fibrous lymph. The omentum is often contracted and greatly thickened. Effusion is usually present, but it varies considerably in amount; it is highly albuminous, and in the tuberculous and cancerous varieties it may be bloody.

Symptoms.—Fever is slight, and may be absent. Pain is not severe, and is commonly paroxysmal. There is usually diffuse tenderness. Anæmia and emaciation may be marked.

Inspection.—The abdomen is generally distended; often irregularly, from sacculated effusions, inflated intestinal coils, or the projecting matted omentum.

Palpation may detect a friction fremitus, and the irregularities noted above. The resistance is often great.

Percussion.—Dulness in the flanks with superincumbent tympany. When the fluid is sacculated, the dulness may be irregularly distributed. Fluctuation can sometimes be elicited.

Prognosis.—Unfavorable.

Treatment.—Rest. Light diet and nutrient tonics (malt, cod-liver oil). Iodide of potassium is given for its absorbent effect. Iodine may be applied externally. When the effusion is great, paracentesis will be required. In the simple and tuberculous forms laparotomy has given encouraging results.

ASCITES.

Definition.—A collection of serous fluid in the peritoneal cavity.

Etiology.—(1) It may result from one of the common causes of dropsy, viz: Bright's disease, chronic heart disease, chronic lung disease, anæmia, and especially cirrhosis of the liver. (2) Pressure of a tumor or displaced viscus upon the portal vein. (3) Chronic peritonitis. (4) Pressure upon the thoracic duct (Chylous ascites).

Symptoms.—When the effusion is large, a sensation of weight, dyspnœa, scanty urine, constipation, and œdema of the feet usually result from pressure.

Physical Signs. Inspection.—The abdomen is distended, the surface is smooth and shining; the base of the thorax is broadened; the navel is more or less obliterated; the superficial veins are frequently enlarged; and, when the patient lies in the dorsal position, the flanks bulge.

Palpation may elicit fluctuation, and in the flanks a sense of resistance.

Percussion.—Dulness and resistance in dependent parts, with superincumbent tympany. Dulness is movable; it is detected in the flanks when the patient occupies the dorsal position.

Aspiration.—The fluid is usually clear, straw-colored, and albuminous; the specific gravity is from 1012–1016.

Diagnosis. *Tympanites, or meteorism.*—This yields universal hyper-resonance on percussion.

Ovarian Cysts.—The enlargement begins in the iliac fossa. The dulness is more or less immovable; as the intestines are pushed aside, there is dulness anteriorly, instead of tympany, as in ascites. Vaginal examination furnishes important data; the fluid has a higher specific gravity and often coagulates spontaneously.

Distention of the Bladder.—The location of the dulness and resistance, the history, and the results of catheterization will render the diagnosis apparent.

Chronic Peritonitis.—In this condition the abdomen is often irregularly enlarged, and the seat of pain and tenderness. Palpation may detect resisting masses. On account of adhesions the dulness may not be movable. The fluid obtained by aspiration is often turbid, contains more than 3 per cent. of albumin, and its specific gravity is above 1015.

Treatment.—When possible, endeavor to remove the cause. Encourage free catharsis by the use of concentrated saline solutions, compound jalap powder (gr. xx–xxx), elaterium (gr. $\frac{1}{8}$). Encourage free diuresis by the use of citrate of caffeine (gr. iij–v), infusion of digitalis (f$\bar{3}$ss), or Niemeyer's pill (page 90).

℞ Potassii citrat., ℥ss ;
Tinct. scillæ, f℥ss ;
Inf. digitalis, f℥iij ;
Aquæ, q. s. ad f℥vj.—M.
Sig.—A tablespoonful thrice daily.

If the effusion is very large, if the stomach is irritable, or if internal remedies fail to give relief, tapping will be required.

DISEASES OF THE PANCREAS.

Pancreatic Hemorrhage.

Etiology.—Slight hemorrhages may be due to venous congestion, the hemorrhagic diathesis, or one of the infectious diseases. The cause of copious hemorrhage (*pancreatic apoplexy*) is obscure. It has been excited by traumatism. It has been observed most frequently in males past forty years of age, in obese subjects, and particularly in those addicted to the free use of alcohol.

Symptoms.—Sudden, severe pain in the epigastrium and the phenomena of collapse are the chief symptoms. Nausea, or vomiting, and tympanites are frequently noted.

Prognosis.—Most cases prove fatal within twenty-four hours, death being due to reflex paralysis of the heart (Zenker). Pancreatitis, cyst of the pancreas, and subphrenic abscess are possible terminations.

Treatment.—This consists in the use of morphine for the relief of pain, and in measures intended to combat collapse.

Acute Pancreatitis.

Varieties.—Hemorrhagic, gangrenous, and suppurative.

Etiology.—Acute pancreatitis often follows pancreatic hemorrhage, and therefore shares in the etiology of the latter. In addition, recurrent gastro-intestinal catarrh is a frequent predisposing cause.

Pathology.—In the hemorrhagic form the organ is irregularly enlarged and the seat of hemorrhagic extravasa-

tion. Opaque, white spots, of a tallowy consistence, are frequently found in the interlobular tissue, omentum, and surrounding parts, and represent areas of *fat necrosis.*

Gangrenous and suppurative pancreatitis are usually secondary to the hemorrhagic variety; in the former there are more or less extensive areas of necrosis, and in the latter single or multiple abscesses. Thrombosis of the portal and splenic veins is frequently encountered.

Symptoms.—The symptoms of hemorrhagic and gangrenous pancreatitis are essentially the same, and consist in severe, deep-seated pain in the epigastrium, vomiting, abdominal distention, collapse, and constipation, followed by more or less fever. Suppurative pancreatitis usually runs a more protracted course, often extending over several weeks, and is characterized by epigastric pain, vomiting, tympanites, chills, and irregular hectic fever.

Diagnosis. *Intestinal Obstruction.*—In this condition the onset is less severe, fecal vomiting is common, and the pain and distention are less frequently limited to the epigastrium.

The history will sometimes serve to differentiate the condition from *biliary colic, perforating gastric ulcer,* and the effects of an *irritant poison.*

Prognosis.—Very unfavorable. The duration varies from a few hours in the severe hemorrhagic forms to several weeks in the chronic suppurative variety.

Treatment.—Palliative. Under favorable circumstances operative interference may be considered.

Chronic Pancreatitis.

Etiology.—It may follow acute pancreatitis. The most common cause is chronic inflammation of the pancreatic duct secondary to gastro-intestinal catarrh. Obstruction of the duct by calculi or tumors may induce it. It may result from syphilis.

Pathology.—The chief lesions are an overgrowth of the fibrous tissue, and atrophy and degeneration of the cellular elements.

SYMPTOMS.—Dyspepsia, diarrhœa, and paroxysms of severe epigastric pain associated with great anxiety and faintness are the most characteristic features. Jaundice is an occasional symptom, and even when it is absent the stools may be light-colored and contain free fat; when the destruction of the gland is extreme glycosuria, with or without the other phenomena of diabetes, is a frequent symptom.

PROGNOSIS.—Serious, and when associated with persistent glycosuria it is almost invariably fatal.

TREATMENT.—The use of fats and starches should be restricted. Carbonated waters are said to increase pancreatic secretion (Becher). Pancreatin or minced pancreas is recommended.

Cancer of the Pancreas.

ETIOLOGY.—The disease most frequently occurs in males past forty years of age.

PATHOLOGY.—Pancreatic cancer is usually primary; it generally involves the head of the gland, and is commonly of the scirrhous variety.

SYMPTOMS.—These include disturbances of digestion, loss of flesh and strength, anemia, deep-seated epigastric pain, and the presence of a tumor. The latter is usually found (50 per cent. of cases) a little above the navel; it is but slightly movable, deep seated, and often pulsatile from its relation to the aorta. The pain often occurs in paroxysms, especially at night, and may be associated with the symptoms of collapse. Jaundice is a frequent symptom, and results from the pressure of the tumor upon the common bile-duct. Pressure on the portal vein may cause ascites. Glycosuria is an occasional symptom. The stools rarely contain free fat, but the presence of abundant undigested muscular fibers in the dejections in the absence of diarrhœa is, according to Fitz, highly suggestive.

DIAGNOSIS.—Gastric cancer. In this condition the tumor is more freely movable, is usually associated with dilatation of the stomach and with marked gastric symptoms. Jaundice is rare.

Cysts of the Pancreas.

Etiology.—The most common cause is obstruction of the duct of Wirsung from stricture, tumor, or impacted calculus. They are occasionally congenital. Traumatism is also a reputed cause.

Pathology.—Pancreatic cysts may be single or multiple. They lie behind the stomach, and may contain from a few ounces to several gallons of a grayish or brownish, viscid fluid, of an alkaline reaction, of a specific gravity between 1010 and 1024, and presenting the characteristics of pancreatic secretion.

Symptoms.—These are very variable, the most common being epigastric pain, vomiting, constipation, or diarrhœa, disturbances of digestion, loss of flesh, and occasionally intestinal hemorrhage. Free fat and much undigested muscular fiber may be found in the stools and sugar in the urine. Physical examination often reveals in the upper part of the abdomen a smooth, elastic, fluctuating tumor which on aspiration yields a fluid capable of emulsifying fats, of converting starch into sugar, and of digesting albumin.

Prognosis and Treatment.—The prognosis is guardedly favorable. Large cysts should receive surgical attention.

Pancreatic Calculi.

Pancreatic calculi are probably due to the retention of secretion from catarrh of the duct, or pressure upon the duct from a tumor or cyst. Their passage through the duct excites *pancreatic colic*, the symptoms of which resemble biliary colic, but the pain is more apt to radiate to the left and is unattended with jaundice. The coexistence of glycosuria, and the discovery in the stools of concretions containing chiefly carbonate or phosphate of lime, would confirm the diagnosis.

DISEASES OF THE LIVER.

The *liver* is situated in the right hypochondrium, with a small part projecting through the epigastrium to the left hypochondrium.

Area of Liver Dulness. — The absolute dulness (part uncovered by lung) extends in the mammary line from the upper border of the sixth rib to the costal margin; in the axillary line, from the eighth rib to the eleventh rib; in the scapular line, from the tenth rib to the eleventh rib; in the median line, the upper border is lost in the cardiac dulness, while the lower border lies midway between the ensiform cartilage and the umbilicus. Slight dulness in the mammary line begins at the fifth rib.

Palpation.

Palpation of the liver is practised to determine position, size, form, and consistence; and to detect any tenderness or pulsation.

Conditions in which the liver is palpable:—

1. In thin subjects, the edge is sometimes palpable under normal conditions.
2. In very young children, in whom the liver is always proportionately large.
3. In depression of the liver, as by a pleural effusion or by a consolidated lung.
4. When the suspensory ligament is relaxed and the liver "wanders."
5. In enlargement from any cause.
6. In certain abnormalities of form, as in the "tight-lace liver."

Superficial Irregularities. — Small irregularities may be noted in cancer, syphilis of the liver, and atrophic cirrhosis.

Large prominences are sometimes noted in tumors, abscesses, and hydatid cysts.

Consistence.—The liver is firm to the touch in hypertrophic cirrhosis, cancer, congestion, and amyloid disease. In abscess

and hydatid disease the resistance is less marked, and sometimes fluctuation can be noted.

Tenderness.—The liver is tender in acute congestion, abscess, cancer, and in affections complicated with perihepatitis.

Pulsation may be detected in the venous congestion resulting from tricuspid regurgitation, abdominal aneurism, in tumors of the left lobe resting on the aorta, and rarely in aortic regurgitation.

Percussion.

Percussion determines size and resistance.

The liver is uniformly enlarged in: (1) Congestion, active and passive. (2) Fatty infiltration. (3) Amyloid infiltration. (4) Hypertrophic cirrhosis. (5) Leukæmic infiltration.

Irregular enlargements of the liver are noted in: (1) Cancer. (2) Abscess. (3) Hydatid disease. (4) Syphilis.

The liver is diminished in size in: (1) Atrophic cirrhosis, late stage. (2) Fatty degeneration. (3) Acute yellow atrophy. (4) Senile atrophy. The area of hepatic dulness may be diminished from certain extrinsic causes, namely, pulmonary emphysema and excessive tympanites.

JAUNDICE OR ICTERUS.

Definition.—Pigmentation of the tissues and secretions with bile-pigments.

Varieties.—(1) Hepatogenous, or obstructive jaundice. (2) Hæmatogenous, or non-obstructive jaundice.

Etiology of Hepatogenous Jaundice.—Obstruction to the outflow of bile leads to its accumulation and re-absorption.

Obstruction may be due to the following causes:—

1. Stricture of the bile-duct, congenital or acquired.
2. Catarrh of the bile-ducts, or of the duodenal mucous membrane around the orifice of the ductus choledochus.
3. Foreign bodies in the ducts; as gall-stones, parasites.
4. Tumors of the liver or of adjacent viscera compressing the

ducts. Fecal accumulations, a pregnant uterus, and displaced organs may similarly compress the ducts.

5. Lowered blood pressure in the vessels of the liver causing increased tension in the bile-ducts, as in the simple icterus of the new-born or that following depressing emotions.

Symptoms.—The skin, mucous membranes, and secretions are stained yellow. The discoloration is usually first noticed in the conjunctivæ. The stools are light, the urine is dark, and in bad cases resembles porter. The pulse is usually slow, and the temperature slightly subnormal. There is always some mental depression, and in extreme cases delirium, convulsions, and coma may develop. Itching of the skin is often noted, and urticaria is a common complication. In grave cases subcutaneous ecchymoses may appear.

Diagnosis.—Other discolorations, like the bronze hue of Addison's disease, and the green tint of chlorosis, must be distinguished from jaundice; but in those cases the conjunctiva is white and the urine lacks bile.

Etiology of Hæmatogenous or Non-obstructive Jaundice.—This form results from a disintegration of the blood, or a destruction of the liver substance. It is sometimes noted in pernicious anæmia, and other grave anæmias, but it more commonly results from the action of some toxic agent on the blood; thus, it may be observed in poisoning by phosphorus, arsenic, and other minerals; in snake-poisoning, in pyæmia, and in certain infectious fevers—as yellow fever, relapsing fever, malarial fever, and acute yellow atrophy.

Symptoms.—Much the same as in obstructive jaundice, but the staining of the skin is usually not so intense, the stools still contain bile, and grave cerebral symptoms are more apt to develop.

ICTERUS NEONATORUM.

Physiological icterus in the newborn is slight, and probably results from the lowered pressure in the portal vessels caused by ligation of the umbilical vein, and the subsequent absorption of bile from the tense capillary ducts.

Pathological icterus in the newborn is marked, and com-

monly proves fatal. It results from congenital stricture of the duct, syphilis of the liver, or septic infection through the umbilical vein.

ACHOLIA.

(Cholæmia, Cholesteræmia.)

This term is applied to a group of symptoms noted in diseases associated with a destruction of the hepatic substance, and probably dependent upon the retention of poisons which should have been eliminated by the liver.

Etiology.—Acholia occurs in acute yellow atrophy, and sometimes at the close of cancer, cirrhosis, and fatty degeneration of the liver.

Symptoms. — Delirium, convulsions, stupor, and coma. Jaundice may or may not be present. Subcutaneous ecchymoses and hemorrhages from mucous membranes are frequently observed.

CATARRHAL JAUNDICE.

(Catarrhal Hepatitis, Catarrh of the Bile-ducts.)

Etiology.—(1) The most common cause is the extension of a gastro-duodenal catarrh into the ducts. (2) Primary inflammation of the ducts may result from exposure to cold and wet. (3) It may be induced by irritation from gall-stones. (4) It may be infectious, complicating malaria, pneumonia, relapsing fever, and similar diseases.

Pathology.—The large ducts are particularly affected; the mucous membrane is swollen and covered with tenacious mucus. When the gall-bladder is compressed, bile is ejected with less ease than is natural through the duodenal orifice. When the catarrhal process is long-continued, ulceration of the ducts, or secondary cirrhosis (biliary cirrhosis) may result.

Symptoms.—(1) Symptoms of gastro-duodenal catarrh usually precede. These are: Coated tongue, anorexia, fetid breath, epigastric distress, vomiting, and perhaps diarrhœa. (2) Obstructive jaundice, indicated by yellow skin and conjunctivæ, light stools, and dark urine. (3) In acute cases,

slight fever and swelling of the liver, which is tender to the touch.

Diagnosis.—Usually easy; the exclusion of other causes of jaundice, and the consideration of the age, acute onset, and preservation of health will usually make the diagnosis apparent.

Prognosis.—Favorable. It rarely becomes chronic and leads to biliary cirrhosis and ulceration of the ducts. The average duration is from a few days to several weeks.

Treatment.—Rest. Liquid diet. Stupes of turpentine or of dilute nitrohydrochloric acid may be applied locally. Mild laxatives are often indicated; calomel may be selected.

℞ Hydrarg. chlor. mit., gr. ij;
Sodii bicarb., ʒj.—M.
Ft. in chart. No. xii.
Sig.—One every hour until a laxative effect is produced.

For the gastro-duodenal catarrh, mineral waters, subnitrate of bismuth (gr. xx), nitrate of silver (gr. $\frac{1}{8}$ q. d.), chloride of ammonium (gr. x, q. d.), phosphate of sodium (ʒj q. d.), are valuable adjuncts. In persistent cases the daily irrigation of the bowel with cold water (1–2 quarts) has been highly recommended; the injections stimulate peristalsis and thus favor the expulsion of mucus and bile from the ducts.

BILIARY CALCULI.

(Gall-stones, Cholelithiasis.)

Definition.—Concretions formed in the gall-bladder, and composed for the most part of bile-elements.

Etiology.—Female sex, age (after forty), heredity, sedentary habits, a rich diet, diseases of the liver which obstruct the flow of bile, as tumors, and catarrh of the ducts.

Pathology.—The stones may be found in the ducts, but they are always formed in the gall-bladder. There may be one or several hundred. When multiple, they are found with facets, from attrition. The size varies from a grain of sand to a large walnut. The color varies from a light yellow to a dark green. The chief constituent is cholesterin, but bile-

acids, bile-pigments, lime, and magnesia also enter into their composition. On section, they usually present a concentric arrangement. The pathogenesis is not known; a chemical change in the bile probably leads to a precipitation of the cholesterin.

Events.—(1) Stones often remain latent in the bladder. (2) They may pass out with pain and spasm (biliary colic). (3) Impaction. A stone may obstruct the cystic duct and lead to distention of the bladder with mucus. More frequently the common duct is obstructed near its duodenal orifice, when the following symptoms result: Permanent jaundice, tenderness, exacerbations of pain, and peculiar paroxysms of fever, chills, and sweats, resembling malaria (Charcot's intermittent). Such paroxysms are not necessarily dependent on suppuration, although abscess may follow obstruction. (4) Perforation into the abdominal sac, stomach, or intestine. External perforation is very rare. (5) After exit, stricture of the duct may result from ulceration, or intestinal obstruction, from impaction.

Symptoms of Biliary Colic.—Sudden and intense pain over the liver, radiating to the back and to the right shoulder. It usually occurs an hour or two after eating. A rigor with fever may mark the onset. The symptoms of intense pain are obvious—anxious face, cold sweat, feeble pulse, and vomiting. Jaundice may follow from obstruction. If the stone escapes, it may be found in the stool.

Diagnosis. *Renal Colic.*—Pain radiates from the kidney down the ureter to the penis; blood in the urine; no jaundice.

Intestinal Colic.—Pain radiates from the umbilicus; flatulence; no jaundice; no stone recovered.

Gastralgia.—Pain referred to stomach and back; no jaundice; no stone recovered.

Prognosis.—The attack usually ends favorably. Recurrence is common. The prognosis, as regards ultimate recovery, should be guardedly favorable; complications are comparatively rare.

Treatment.—*The Attack.*—Hot fomentations. Morphine (gr. $\frac{1}{8}$ to $\frac{1}{2}$) with atropine (gr. $\frac{1}{100}$) hypodermically. In aggravated cases anæsthetics will be required.

The Interval.—A regulated diet, largely vegetable. Systematic exercise should be enjoined. The flow of bile should be encouraged by the use of mineral waters, phosphate of sodium, or a vegetable cholagogue, like podophyllin or euonymin. Catarrh of ducts should be relieved so that stones may escape.

In impaction the same treatment is indicated with counter-irritation, and the use of some intestinal antiseptic, such as salol, naphthol, or the salicylate of bismuth, to replace the antiseptic elements of the bile.

In aggravated cases an exploratory incision should be made, when a stone may be removed from the common duct (choledochotomy), or from the gall-bladder (cholecystotomy), or the gall-bladder removed (cholecystectomy).

ACUTE INFLAMMATION OF THE GALL-BLADDER.

(**Acute Cholecystitis.**)

Etiology.—It is frequently dependent upon the presence of gall-stones. It may follow (sometimes remotely) one of the acute infections, notably typhoid fever. Distention of the gall-bladder from catarrhal swelling of the cystic duct is a possible cause. The micro-organisms most frequently found in the pus are the colon bacillus, typhoid bacillus, and diplococcus of pneumonia.

Pathology.—The gall-bladder is more or less distended, conical in shape and surrounded by adhesions. Its walls are opaque, granular, and changed in color. Its contents are dark, puriform or hemorrhagic. Its mucous membrane is deeply injected. Left to itself, suppurative cholecystitis may eventuate in rupture or gangrene.

Symptoms.—Severe paroxysmal pain in the right hypochondriac region; irregular fever with its attending phenomena; nausea or vomiting; and locally, more or less circumscribed tenderness with rigidity, and in some cases an elastic tumor attached to the liver; chills and jaundice are occasional symptoms.

Diagnosis.—It must be distinguished from *appendicitis*,

subphrenic abscess, *pancreatic cyst*, and *pancreatic hemorrhage*. The discriminating features are the locality of the pain and tenderness, and the seat of the tumor.

PROGNOSIS.—Grave in suppurative cases. Of 82 cases, 53 proved fatal (Da Costa). Early operation, however, offers considerable hope of success.

TREATMENT.—This consists in incision and free drainage.

HYPERÆMIA OF THE LIVER

VARIETIES.—(1) Active hyperæmia. (2) Passive hyperæmia.

ETIOLOGY.—*Active hyperæmia* is commonly due to dietetic indiscretions (biliousness). It may result from over-indulgence in alcohol. It is often present in the infectious fevers. It appears to arise idiopathically in hot climates.

Passive hyperæmia results from diseases which obstruct the venous circulation, as chronic heart and lung disease.

PATHOLOGY.—The liver is enlarged and filled with blood. In the passive variety, the centre of the lobule, the area of the hepatic vein, is deeply pigmented, while the periphery, the area of the portal vein, is pale. This mottled appearance has given rise to the term "nutmeg liver." In persistent cases, pigmentation, atrophy of liver-cells, and overgrowth of connective tissue result—a condition termed "cyanotic induration."

SYMPTOMS. *Active hyperæmia.*—It is associated with gastric catarrh, and the usual symptoms are: Coated tongue, fetid breath, anorexia, pain and tenderness in the epigastric and hypochondriac regions, nausea, vomiting, sick-headache, and sometimes slight jaundice. The liver may be enlarged

In the *passive variety*, the symptoms are the same, though less marked. The liver is often quite large, and in extreme cases, such as follow tricuspid regurgitation, it may pulsate.

PROGNOSIS.—In simple active congestion the prognosis is good. In passive congestion the prognosis depends on the cause.

TREATMENT. *Active hyperæmia from dietetic errors*—Restrict the diet, apply counter-irritants, and administer calomel and soda, thus :—

℞ Hydrarg. chlor. mit., gr. j ;
Sodii bicarb., ʒj.—M.
Ft. in chart. No. vi.
Sig.—One every hour until three or four have been taken.

Follow the calomel with a laxative dose of sodium phosphate, Carlsbad or Rochelle salts.

In recurring attacks of biliousness, in addition to dietetic and hygienic directions, the following will prove useful :—

℞ Mass. hydrarg., gr. v ;
Pulv. rhei,
Ext. gentian., āā ʒss ;
Ol. caryophyll. gtt. iv.—M. (HARTSHORNE.)
Div. in pil. No. xx.
Sig.—One or two occasionally, as directed ; to be continued if required, thrice daily for several days.

In passive congestion, direct the treatment to the original disease. In mild cases the mineral waters do well (Carlsbad, Congress, and Friedrichshall). A mercurial laxative may be used from time to time. In obstinate cases the concentrated salines may be employed as purgatives, and wet cups applied to the liver.

CIRRHOSIS OF THE LIVER.

(Hob-nailed Liver, Interstitial Hepatitis, Gin-drinker's Liver.)

DEFINITION.—A chronic disease characterized anatomically by a hyperplasia of the connective tissue and more or less destruction of the secreting cells.

ETIOLOGY.—Male sex and middle life are generally predisposing factors. (1) The abuse of spirituous liquors is a common cause. (2) It follows chronic diseases which alter the crasis of the blood, viz : Syphilis, gout, malaria, and tuberculosis. (3) It results from the passive congestion induced by chronic heart and lung disease. (4) It may be secondary to inflammation of the bile-ducts. It is sometimes seen in

children; and in them, congenital syphilis and the infectious fevers appear to be the exciting causes.

Pathology.—Two varieties have been recognized: (1) Atrophic cirrhosis, and (2) hypertrophic cirrhosis.

Atrophic Cirrhosis.—In the early stages the liver is somewhat large from hyperæmia. In the advanced stage the liver is small, firm, gray in color, and covered with numerous granulations ("hob-nails"). A section of the liver presents a network of fine and of coarse pearly bands of connective tissue. The contraction of this connective tissue is responsible for the reduction in size and granular surface.

Microscopic examination reveals an overgrowth of connective tissue, of a fibrous character, and chiefly perilobular in distribution. The contraction of this tissue constricts the branches of the portal vein and causes atrophy and degeneration of the liver cells.

The term *hypertrophic cirrhosis* is applied to a condition in which the connective-tissue hyperplasia starts from the periphery of the capillary bile-ducts, instead of from the ramifications of the portal vein, as in atrophic cirrhosis. The symptoms of portal obstruction are not marked, but jaundice is a prominent feature.

The liver is large, yellow in color, and its surface is smooth or finely granular. The increased size is due to a great overgrowth of connective tissue, and to preservation of the hepatic parenchyma.

The connective tissue is not fibrous, but embryonal (round-cell infiltration), and therefore does not contract. Its presence, however, between the liver cells leads to compression of the biliary capillaries.

Symptoms of Atrophic Cirrhosis.—Obstruction to the portal circulation induces congestion of the stomach and intestines, and hence the initial symptoms are those of gastro-intestinal catarrh. These are: Coated tongue, anorexia, fulness and distress after eating, vomiting frothy mucus, flatulence, constipation, and dark urine. These phenomena may last for months or years.

As the obstruction becomes greater the, portal blood finds new channels, and the superficial abdominal veins enlarge,

notably around the umbilicus, forming the so-called "caput medusæ." Hemorrhoids result from the same cause.

Engorgement of the portal system leads to ascites and swelling of the feet, to hemorrhage from the stomach, bowel, or some distant organ, and to enlargement of the spleen.

The liver is at first large, but is subsequently contracted.

There is loss of flesh and strength. The skin is muddy in appearance. Jaundice is not common, and when present results from catarrh of the bile-ducts. Death results from exhaustion, hemorrhage, intercurrent disease, or from a group of cerebral symptoms (delirium, convulsions, and coma) which are probably due to the retention of some toxic agent which the liver should eliminate.

Hypertrophic Cirrhosis.—Jaundice is marked. The liver is enlarged, smooth, and firm. Symptoms of portal obstruction, such as dropsy and hemorrhages, are not common. The spleen is swollen. Moderate fever and leucocytosis are generally present, and favor the view that the disease is of an infectious nature. The disease may last one or two years, but an abrupt termination in convulsions and coma may occur at any time.

Complications.—Tuberculosis, interstitial nephritis, cardiac hypertrophy, and hemorrhage.

Diagnosis.—In the early stage the diagnosis can only be suspected. In the drunkard, chronic gastric catarrh with enlargement of the liver would strongly indicate cirrhosis.

Cancer.—History, greater cachexia, jaundice more common, and ascites less frequent, liver enlarged and studded with nodules, other organs affected, pain, and short duration.

Chronic Peritonitis with effusion.—This is usually tuberculous or cancerous. The short duration, the abdominal tenderness, the lack of a uniform enlargement from bands of lymph, the absence of symptoms indicating portal obstruction, the normal size of the liver, after tapping, and the turbid sanious fluid will indicate chronic peritonitis.

Prognosis.—Unfavorable. It may be arrested in the early stage. The entire duration may be many years, but death usually results in from one to three years after symptoms of portal obstruction have appeared.

Treatment.—Light nutritous diet. Rest. Alcohol must be interdicted. Treat the gastric catarrh with nitrate of silver, bismuth, mineral waters, and antiseptics (creosote or salicylate of bismuth). Iodide of potassium in small doses, well diluted, may be of service in the early stage. Counter-irritation over the liver should be frequently practised.

Ascites.—Concentrated saline purges in the morning (Epsom salts ℥ss in enough water to dissolve it). Diuretics, as digitalis or caffeine. Niemeyer's pill may be useful:

℞ Mass. hydrarg., gr. xij;
Pulv. digitalis, gr. xij;
Pulv. scillæ, gr. xij.—M.
Ft. in pil. No. xii.
Sig.—One pill thrice daily.

When the effusion is very large, internal remedies fail, and paracentesis will be required.

The Operation.—Empty the bladder. Anæsthetize a point in the linea alba midway between the umbilicus and pubis. Tap with a small trocar, and have a long rubber tube attached to the canula for conveying the liquid into a convenient receptacle. When the liquid stops flowing withdraw the canula, cover the wound with adhesive plaster, and apply an abdominal binder. Observe strict antisepsis. The operation is free from danger.

ABSCESS OF THE LIVER.

Etiology.—(1) The presence in the liver of the amœba coli of dysentery. (2) Traumatism. (3) Foreign bodies, gall-stones, retained bile, and hydatid cysts. (4) Septic emboli; they may come through the hepatic artery, but usually they come through the portal vein from gastric ulcers, or the ulcers of dysentery, typhlitis, or typhoid fever, and produce a purulent inflammation of the vein (suppurative pylephlebitis).

Pathology.—The abscess following amœbic dysentery is often single, and usually occupies the right lobe.

Embolic abscesses are always multiple.

Events.—Hepatic abscess may kill by exhaustion or by rupture into adjacent viscera. Recovery may follow after

operation or spontaneous evacuation; and the latter may be external, through the bronchial tubes, or through the bowel.

Symptoms.—Hectic symptoms: Fever, high in the evening and low in the morning, sweats, and chills. Local symptoms: The liver is enlarged, painful, and tender. There may be bulging and even fluctuation. Pus may be detected by the aspirating needle. Jaundice from obstruction is sometimes present.

Diagnosis. *Hydatid Cysts.*—Long duration, history, clear fluid on aspiration, absence of pain, and absence of hectic symptoms.

Cancer.—History, cachexia, the involvement of other organs, multiple and firm nodules, and absence of hectic symptoms.

Intermittent Fever due to Impacted Calculi.—Fever and pain are periodic; the health may be well preserved; the liver is not enlarged. The condition may persist for several years.

Prognosis.—Embolic abscesses (multiple) prove invariably fatal. Traumatic abscesses or abscesses due to a amœbic dysentery may terminate favorably after spontaneous or induced evacuation.

Treatment.—This consists in evacuation of the pus and the establishment of thorough drainage.

CANCER OF THE LIVER.

Etiology.—Male sex, age (after forty), heredity, traumatism, and obstruction from gall-stones are predisposing factors.

Pathology.—It is generally secondary. The liver is enlarged, and studded with numerous grayish-white nodes, some of which project from the surface. The superficial nodes are often depressed at the centre.

Symptoms.—(1) Severe pain and tenderness. (2) Cachexia, *i. e.* loss of flesh and strength, with pallor. (3) Pressure-symptoms: jaundice is common but ascites is rare. (4) Physical examination: the liver is enlarged, its surface is nodular, and the central depression, or umbilications, can often be detected. (5) Symptoms of the primary growth which is usually in the stomach.

Fever is generaly absent, but secondary perihepatitis or suppuration of the cancerous nodules may induce it.

Diagnosis. *Hypertrophic Cirrhosis.*—Liver is smooth and painless, the duration is longer, cachexia is not marked, and there is no indication of a primary cancer.

Hydatid Cysts.—Health preserved, tumor elastic or fluctuating, no pain, jaundice uncommon, aspiration yields a clear fluid containing hooklets.

Abscess.—History, short duration, hectic fever, and results of aspiration.

Prognosis. — Absolutely fatal. Duration, from a few months to a year.

Treatment.—Palliative.

AMYLOID LIVER.

(Waxy Liver, Lardaceous Liver.)

Definition.—An enlargement of the liver due to the deposition of an albuminoid substance.

Etiology.—(1) Prolonged suppuration; (2) syphilis; (3) tuberculosis, and (4) chronic malaria are causal factors.

Pathology.—The liver is very large, hard, and smooth. The edge is blunt. On section, the surface is "waxy," and a dilute solution of iodine strikes a mahogany-red color with the amyloid material. The degenerative process begins in the walls of the capillaries and spreads to the connective tissue.

Symptoms.—Failure of general health with anæmia. The liver is enlarged, smooth, firm, and painless, and presents a blunt edge. The spleen and kidneys share in the degeneration, and, as a result, the spleen is enlarged and the urine is albuminous.

Diagnosis.—The history, the smooth, painless, enlargement of the liver without jaundice, and the involvement of the kidneys and spleen, are the diagnostic phenomena.

Prognosis.—Unfavorable.

Treatment.—Remedies must be directed to the causal disease. Nutrients and tonics are indicated. Absorbents, like the iodides, mercurials, and ammonium chloride, have been recommended, but are valueless.

HYDATID CYSTS OF THE LIVER.

(Echinococcus of the Liver.)

Etiology and Pathology.—Hydatid cysts are formed by the embryos of the tænia echinococcus, a small tape-worm inhabiting the intestines of the dog.

The eggs of the worm are accidentally ingested by man, and embryos are liberated in the stomach, whence they may migrate to any organ; the liver however is most commonly affected through the portal vein. The fixed embryo soon develops into a cyst which is composed of an external laminated layer and an internal breeding layer. A connective-tissue layer is formed on the outside from irritation.

The cyst contains a clear non-albuminous fluid which has a specific gravity of 1005 to 1007, and which is rich in chlorides.

Scolices or larvæ develop from the breeding layer; they are provided with four suckers and a circle of hooklets, and produce daughter-cysts within the parent-cyst. When ingested by the dog the larvæ develop into mature tape-worms.

Symptoms.—Small cysts excite no symptoms. There is often a slowly-developing, irregular enlargement of the liver; if the cyst is superficial, an elastic or fluctuating mass may be detected on palpation.

On percussion a peculiar vibratory sensation (hydatid thrill) may be imparted to the hand. Aspiration yields a clear fluid containing hooklets.

Fever, pain, and jaundice are usually absent.

Events.—(1) It may reach a certain size, and then remain latent. (2) Trifling injury may convert it into an abscess. (3) Rupture of the cyst externally or into neighboring organs may result in death or in recovery.

Diagnosis.—Slow development, irregular enlargement, elastic feel, the results of aspiration, and the absence of pain, fever, and jaundice are the diagnostic features. Suppurating cysts will be regarded as abscesses. An upward-growing cyst may present the signs of a pleural effusion.

Prognosis.—Guardedly favorable.

Treatment.—This consists in incision, evacuation, and drainage.

ACUTE YELLOW ATROPHY.

(Acute Parenchymatous Hepatitis; Malignant Jaundice.)

Definition.—A rare and grave disease characterized anatomically by a rapid destruction of the liver tissue, and manifested by jaundice, hemorrhages, a reduction in the size of the liver, and marked cerebral phenomena.

Etiology.—Female sex, pregnancy, early life, are predisposing factors.

Alcoholic excesses, emotional excitement, and syphilis have been given as exciting causes.

The rapid course, widespread lesions, and the fact that it has occurred endemically suggest an infectious origin.

Pathology.—From destruction of its substance the liver is quite small. The capsule, being too large for the shrunken organ, is wrinkled. The surface is yellowish-red and mottled.

Histology.—Fat drops, molecular débris, fat crystals, and crystals of leucin and tyrosin take the place of normal liver-cells. The other organs reveal fatty degeneration.

Symptoms.—(1) The initial symptoms, which are those of catarrhal jaundice, are: Malaise, slight fever, coated tongue, nausea, vomiting, and jaundice. (2) Nervous symptoms follow; these are: Severe headache, delirium, convulsions, and coma. Sometimes these symptoms precede the jaundice. (3) The urine is scanty, and contains albumin, blood, tube-casts, and crystals of leucin and tyrosin. (4) Hemorrhages are common, the skin may be covered with ecchymoses, and bleeding from the mucous membranes may occur. (5) The area of hepatic dulness is diminished, but the area of splenic dulness is increased.

Diagnosis.—The grave cerebral symptoms, reduced hepatic dulness, and hemorrhages will separate it from *catarrhal jaundice.*

Phosphorus-poisoning.—History, phosphorus in the urine, primary enlargement of the liver, and the great severity of the initial gastric symptoms.

Prognosis—Almost invariably fatal. Death results within a week after the appearance of cerebral symptoms.

Treatment.—Palliative.

DISEASES

OF

THE KIDNEYS.

THE URINE.

Normal urine is a pale, amber-colored fluid, of acid reaction, having a specific gravity of 1015 to 1025, and amounting in quantity to about fifty ounces in twenty-four hours.

Polyuria.—An increased flow of urine.

Temporary polyuria results from: (1) Excessive ingestion of fluids. (2) Diuretics. (3) Suppression of perspiration. (4) Crises of certain febrile diseases, and certain neurotic manifestations, such as excitement, neuralgia, and hysteria. (5) Abdominal enlargements, as in pregnancy, effusions, and tumors. (6) Removal of some temporary obstruction in the urinary passages.

Permanent polyuria results from: (1) Diabetes mellitus. (2) Diabetes insipidus. (3) Chronic interstitial nephritis. (4) Amyloid kidney.

The **urine is diminished or suppressed** (anuria) in the following conditions: (1) Excessive secretion through other channels, as in free perspiration and diarrhœa. (2) In fever. (3) Passive renal congestion, from obstructive heart, lung, or liver disease. (4) Organic obstruction in the urinary passages. (5) In acute and chronic parenchymatous nephritis. (6) Nervous causes, as in hysteria, and in the reflex inhibition after abdominal injuries or operations.

Urea.—Urea results from the perfect decomposition of the nitrogenous elements of food and tissues. It is perfectly solu-

ble in urine, but the nitrate of urea crystallizes in the form of transparent imbricated plates when nitric acid is added to urine that has been partially evaporated.

The amount of urea excreted varies greatly in health. Normal urine contains about 2 to 2½ per cent. of urea.

It *is increased:* (1) After the ingestion of much albuminous food. (2) After exertion. (3) In acute inflammatory processes and in fevers. (4) In diabetes.

It *is diminished:* (1) In nephritis. (2) In organic diseases of the liver. (3) In wasting diseases and in anæmia. (4) In starvation.

Fowler's Hypochlorite Test for Urea.—Add to 1 volume of the urine 7 volumes of Labarraque's solution of chlorinated soda. Shake the jar containing the mixture occasionally, and stand it aside for two hours, when the urea will have been decomposed. Now take the specific gravity of the quiescent fluid.

2d. Ascertain the specific gravity of the mixture of urine and Labarraque's solution before decomposition. To do this, multiply the specific gravity of the pure Labarraque's solution by 7, add this to the specific gravity of the pure urine, and divide by 8. The result is the specific gravity of the mixed fluid. From this subtract the specific gravity of the quiescent mixture after decomposition of the urea, multiply the difference by .77, and the result is the percentage of urea.—TYSON.

Lithuria.—Uric acid or urates in the urine. These substances are formed by the imperfect metamorphosis of tissues and nitrogenous food. When they are in excess the urine is heavy, dark in color, and on cooling throws down a brick-red deposit, termed "lateritious" (*later*, a brick).

Microscopically, uric acid appears as reddish-yellow rhombic prisms or lozenge-shaped crystals.

Amorphous urates appear as fine, dark, and opaque granules.

Crystalline urates appear as needles, dumb-bells, or as globular masses from which sharp spines project.

Murexide Test for Uric Acid and its Salts.—Evaporate a little urine in a porcelain dish, add a drop or two of strong nitric acid, and heat again to dryness. Cool, and add a drop of

liquor ammoniæ, and the beautiful purple color of murexide is developed.

Fig. 3.

Uric acid and uric acid salts.

Urates.—The urates are present in small quantity in normal urine. They may become perceptible or transiently increased: (1) In urine exposed to a cold atmosphere. (2) In urine made scanty by free perspiration or diarrhœa. (3) When the acidity of the urine is temporarily increased. (4) After the excessive indulgence in nitrogenous food.

The urates are increased pathologically in many diseases which directly or indirectly interfere with tissue or food metabolism, notably in: (1) Lithæmia or the gouty diathesis. (2) Fever. (3) Extreme anæmia, (4) Diseases of the lungs—from interference with oxidation.

Leucin and Tyrosin.—These substances are found in the urine in certain specific fevers, in grave anæmia, and especially in fatty degeneration of the liver resulting either from phosphorus-poisoning or acute yellow atrophy.

They may be detected by evaporating a few drops of the urine on a glass slide. Leucin appears in the form of small, round, glistening spheres, resembling fat drops, but unlike the latter they are insoluble in ether. Tyrosin appears in the form of intersecting tufts of fine acicular crystals.

Fig. 4.

a. Tyrosin crystals. *b.* Leucin crystals.

Phosphates.—There are two forms, amorphous and crystalline.

Amorphous earthy phosphates are found in alkaline urine, and are precipitated by adding a few drops of liquor ammoniæ to the urine.

Crystallized phosphate of lime appears as stellar or rod-shaped crystals which are soluble in acetic acid.

Fig. 5.

Triple phosphate.

The ammonio-magnesian phosphate, or triple phosphate, appears in decomposing urine as transparent coffin-shaped prisms. They may resemble crystals of oxalate of lime, but, unlike the latter, are freely soluble in acetic acid.

The presence of phosphates in the urine is no indication of excess, for when normal in amount they are often precipitated in urine that is temporarily alkaline.

The detection of triple phosphates in newly-voided urine indicates decomposition in the bladder, a condition resulting from vesical catarrh.

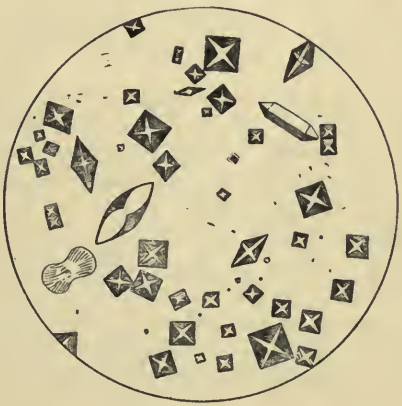
Phosphates are often increased in nervous dyspepsia, melancholia, and neurasthenia.

Chlorides.—The quantity of these salts is increased: (1) After exertion. (2) During the absorption of mechanical or inflammatory effusions. (3) In intermittent fever, from the destruction of corpuscles.

The quantity is decreased: (1) In most febrile diseases. (2) In nephritis. (3) In many wasting diseases. (4) Especially in pneumonia.

Test.—We may thus roughly estimate the quantity. Add a few drops of strong nitric acid to the urine, remove any albumin that may be present, and then add to the clear urine a little of a strong solution of nitrate of silver. The abundance of the white precipitate will indicate the quantity of chlorides present.

Fig. 6.



Oxalate of lime.

Oxaluria.—Oxalate of lime appears in the urine as dumb-bell-shaped crystals, or as minute highly refracting octahedra.

Many conditions produce them. They are found: (1) After eating certain fruits and vegetables, as rhubarb, cauliflower, and pears. (2) In certain diseases, notably nervous dyspepsia, hypochondria, melancholia, diabetes, and wasting diseases.

In these cases the oxalates result from the imperfect metabolism of organic substances.

Urobilinuria.—Urobilin is a coloring principle derived from the blood. When present in the urine in large amount it produces a reddish-brown color; when deposited in the tissues it produces a form of jaundice which has been called urobilin-icterus (Jaksch).

Urobilinuria occurs: (1) Occasionally in health. (2) In pyrexia. (3) After the absorption of hemorrhagic effusions. (4) In liver disease. (5) In grave anæmia.

Glucosuria, or Glycosuria.—Glucose in the urine.

Its Causes.—(1) Normal urine contains a trace. (2) Diabetes mellitus. (3) Certain diseases, as gout, chorea, tetanus, and functional nervous affections. (4) Ingestion of much saccharine material. (5) Pregnancy. (6) Toxic substances in the blood, as the nitrites, phloridzin, and carbon monoxide. (7) Lesions of the pancreas, liver, and base of the brain.

Qualitative Tests for Glucose.—The copper tests are commonly employed, and depend on the power which glucose possesses of converting blue oxide of copper into the orange-yellow suboxide.

Trommer's Test.—Add to the suspected urine half its volume of liquor potassæ, and if any precipitate falls filter the solution; then add one or two drops of a weak solution (1–30) of sulphate of copper, and heat the resulting mixture. If sugar is present, a dense yellow or red precipitate falls.

Simple decolorization of the fluid is no proof of sugar.

Fehling's Test.—As the fluid employed in this test spoils on keeping, it should be freshly prepared when required by mixing in equal proportions the following solutions:—

First solution: Dissolve 34.64 grams of pure cupric sulphate in distilled water, and dilute up to 500 cubic centimeters.

Second solution: Dissolve 180 grams of pure Rochelle salt and 70 grams of caustic soda in 400 cubic centimeters of dis-

tilled water, and heat to boiling; on cooling, make up to 500 cubic centimeters with distilled water.

To about ten minims of each solution in a test-tube add about a fluid drachm of distilled water, and boil for a few seconds; if the solution remains clear, add the suspected urine drop by drop, and occasionally heat the tube. If sugar is abundant, a yellowish-red deposit will be produced. If no precipitate falls, continue the addition of the urine until an equal volume has been added, and allow to cool; then if no precipitate falls, sugar is absent.

The Phenyl-hydrazin Test.—Put in a test-tube half filled with water phenyl hydrazin (hydrochlorate) 2 grains and sodium acetate 3 grains. Dissolve by heating. Fill the tube with suspected urine, and stand in boiling water for twenty minutes. Then place in cold water. On cooling yellow radiating groups of needle-shaped crystals of phenyl-glucosazon fall, which may be detected under the microscope.

Böttger's Test.—Add to a couple of drachms of suspected urine which is free from albumin an equal volume of liquor potassæ and a few grains of subnitrate of bismuth, and boil; if sugar is present, it will reduce the salt of bismuth to black metallic bismuth. Substances containing sulphur, like albumin, yield a similar black precipitate.

The Fermentation Test.—Fill a four-ounce bottle three parts full of urine, and add a fluid drachm of ordinary yeast, or a small portion of compressed yeast, lightly cork, and subject to a temperature of 70° to 80° Fahr. for ten or twelve hours. If sugar is present, fermentation results with the evolution of carbon dioxide, and the specific gravity of the urine falls.

Quantitative Tests.—Fermentation test: Employ two bottles of urine, and to the one add the yeast; at the end of twenty-four hours take the specific gravity of each specimen. Every degree lost in the fermented urine indicates a grain of sugar to the fluidounce.

Fehling's Test.—To one cubic centimetre of Fehling's solution add four cubic centimetres of distilled water, and boil; if the solution still remains clear, add $\frac{1}{10}$ c. c. of the urine from a graduated pipette, and gently heat. Continue the addition of the urine, little by little, until all blue color has dis-

appeared. If one cubic centimetre of urine has been added, it will have contained half of one per cent. of sugar. If two c. c. are used, it will have contained one-quarter per cent. If but a half of a cubic centimetre is used, it will have contained one per cent.

If the specific gravity indicates that the amount of sugar is great, dilute the urine with a definite amount of water, and estimate accordingly (Tyson).

Albuminuria.—Albumin in the urine.

Its Causes.—(1) All forms of nephritis. (2) Congestion of the kidney, as the result of chronic heart, lung, or liver disease. (3) Pregnancy. (4) Cyclical. The urine may be albuminous at certain times, as after meals, heavy exercise, bathing, or on rising in the morning. (5) Accidental. From the admixture of albuminous substances with the urine, as pus, semen, and blood. (6) Certain nervous diseases, as epilepsy, tetanus, and injury to the brain. (7) Extreme anæmia. (8) Ingestion of large amounts of albuminous food.

Tests for Albumin. Heller's Test.—Pour a small quantity of colorless nitric acid in a test-tube, and allow an equal quantity of filtered urine to trickle from a pipette down the sides of the tube and to come in contact with the acid. If albumin is present, a sharply-defined white ring is formed at the line of junction.

Turpentine, copaiba, and other oleoresins eliminated in the urine yield similar rings, but the latter are redissolved on the addition of alcohol.

Uric acid produces an undefined pink ring, but it is not exactly at the line of contact, and is redissolved on the application of heat.

Johnson's Test.—Fill a six-inch test-tube two-thirds full of filtered urine, and allow a couple of drachms of a clear saturated solution of picric acid to flow down the side of the tube and to mix with the urine. Turbidity indicates the presence of albumin, and it increases on gently heating the tube near its mouth. Certain substances in the urine, like the alkaloids, produce a similar turbidity, but this disappears on the application of heat.

Roberts's Nitric Magnesian Test.—Very delicate and reliable

The test-fluid is made by adding one volume of strong nitric acid to five volumes of a saturated solution of sulphate of magnesium, and is employed in the same manner as nitric acid in Heller's test.

Acetonuria.—Acetone results from the metamorphosis of albumin, and is found in the urine in many conditions, notably: (1) A trace in normal urine. (2) In Cancer. (3) Febrile diseases. (4) Psychoses. (5) It may arise as a primary condition (Von Jaksch). (6) In diabetes it is often abundant. (7) After operations.

Legal's Acetone Test.—To four c.c. of urine, rendered alkaline with liquor potassæ, add a few drops of a strong solution of sodium nitro-prusside. If the red color produced turns purple on the addition of a few drops of concentrated acetic acid, acetone is present.

Diaceturia and Oxybuturia.—Diacetic acid and oxybutyric acid are never found in normal urine, but are found associated with acetone in certain fevers, and especially in diabetes. Their decomposition yields acetone, and they are probably the cause of diabetic coma.

Test for Diacetic Acid.—Boil the urine and add a solution of ferric chloride. If diacetic acid is present, a Burgundy-red color develops.

Hæmaturia.—Blood in the urine.

The chief causal conditions are: (1) Vicarious menstruation. (2) Traumatism applied to any part of the genito-urinary tract. (3) General blood dyscrasia, as in the specific fevers, purpura, malaria, scurvy, etc. (4) Congestion of the kidney from chronic heart, lung, or liver disease. (5) Acute inflammation of any part of the genito-urinary tract. (6) Stone in the genito-urinary tract. (7) Varicose veins at the neck of the bladder. (8) It may occur paroxysmally without obvious cause. (9) Parasites in the genito-urinary tract, as the Filaria sanguinis hominis and the Distoma hæmatobium. (10) Tumors and tubercle of the kidney and bladder.

Diagnosis.—By the color of the urine and by microscopic and spectroscopic examination.

Heller's Test.—Boil the urine with a solution of caustic potash: phosphates are precipitated, which assume a red color from the freed hæmatin.

Source of the Hemorrhage. Urethra.—The urine first passed is bloody, and the other symptoms point to the urethra.

Bladder. — Bleeding often at the end of micturition, and other symptoms, point to the bladder.

Kidney.—Blood intimately mixed. There may be blood-casts or clots, and the other symptoms point to the kidneys.

Hæmoglobinuria.—Blood-pigment in the urine.

The chief causal conditions are: (1) Blood disintegration from the specific fevers, scurvy, purpura, malaria, etc. (2) Absorption of internal hemorrhagic effusions. (3) It follows transfusion of blood. (4) Paroxysmally, without obvious cause. (5) Poisons, such as carbolic acid, potassium chlorate, phosphorus, etc.

Indicanuria.—Indican is a colorless compound resulting from the decomposition of albuminous substances in the small intestine, and by oxidation is converted into indigo.

It occurs (1) Frequently in health. (2) From undue retention of material in the small intestine, as in peritonitis, intestinal obstruction, and obstinate constipation. (3) In wasting diseases. (4) Purulent inflammations. (5) Asiatic cholera.

Test for Indican.—Mix equal volumes of urine and fresh hydrochloric acid, and add, drop by drop, a fresh concentrated solution of chloride of lime (5 to 1000). Indican is indicated by the appearance of an indigo-blue color.

Choluria.—Bile in the urine. *Bile-pigment* is found in the urine in all forms of jaundice.

Bile-acids in the urine indicate hepatogenous jaundice, but their absence in jaundice is no proof that the latter is hæmatogenous in origin.

Gmellin's Test for Bile-pigment.—Allow a few drops of urine and a few drops of fuming nitric acid to come together on a white plate. If bile is present, there will be an iridescent play of colors—green, blue, violet, and red—at the line of contact.

Pettenkoffer's Test for Bile-acids.—Add a few grains of cane-sugar and a drop of sulphuric acid to the suspected urine in a test-tube; heat gently, and if bile-acids are present a violet-red color is produced.

Chyluria.—Chyle in the urine. It produces a milky turbidity which gradually rises to the top of the urine in the form

of pellicles of finely-divided fat. Its chief causes are: (1) Injury to the lymphatic ducts. (2) Pregnancy. (3) Obstruction of the lymphatic ducts by the Filaria sanguinis hominis, a thread-worm most commonly met with in the tropics.

Pyuria.—Pus in the urine. It results (1) from suppurative inflammation of any part of the genito-urinary tract, and (2) from the rupture of abscesses into the tract.

It appears as a dull, greenish-yellow precipitate which is converted into a clear gelatinous mass by the addition of liquor potassæ. It can always be detected by the microscope.

Source.—When pus is from the kidney it is intimately mixed with the urine, the latter has an acid or neutral reaction, and the associated symptoms point to the kidneys.

When the pus is from the bladder it is not so intimately mixed with the urine; the latter is usually alkaline in reaction, and the associated symptoms point to the bladder.

RENAL HYPERÆMIA.

Varieties.—(1) Active hyperæmia, and (2) passive hyperæmia.

Active Hyperæmia.

(Acute Congestion.)

Causes.—(1) Exposure to cold when the body is overheated. (2) Eruptive fevers. (3) Poisons, as the stimulating diuretics. (4) Pregnancy.

The same cause aggravated would produce acute nephritis.

Pathology.—The kidney is swollen, of a deep red color, and bleeds freely on section. Microscopic examination reveals cloudy swelling of the renal epithelium.

Symptoms.—Pain over the loins. The urine is dark, scanty, of high specific gravity, and may contain a trace of albumin, a few hyaline casts, and some free blood.

Prognosis.—If the cause can be removed, the prognosis is favorable.

Treatment.—Absolute rest. Wet cups or warm fomentations over the loins. Liberal use of water. Saline laxatives. Encourage sweating by the vapor bath or small doses of pilo-

carpine. The infusion of digitalis may be used to increase the quantity of urine.

Passive Hyperæmia.

(Chronic Congestion.)

Etiology.—(1) Causes which obstruct the general circulation, as chronic heart, lung, and liver disease. (2) Pressure of tumors on the renal veins. (3) Rarely thrombosis of the renal veins.

Pathology.—The kidney is swollen and of a bluish-red color, and later becomes hard from an overgrowth of connective tissue (cyanotic induration). In advanced cases the renal epithelium is fatty.

Symptoms.—Sensation of weight over the loins. The urine is usually diminished, but is rarely increased in quantity. Free blood, a little albumin, and occasionally a few narrow hyaline casts are found.

Diagnosis.—The comparative absence of albumin and casts, the absence of dropsy and uræmic symptoms, and the presence of urea in normal amount will separate congestion from nephritis.

Prognosis.—Depends on the cause.

Treatment.—Rest. Light diet. Dry cups to the loins. The use of diuretics when the urine is scanty. The following tonic diuretic pill may be of service:—

℞ Quininæ sulph., gr. xxx;
Pulv. digitalis, gr. xxx;
Pulv. scillæ, gr. xxx;
Ext. nucis vomicæ, gr. v;
Pulv. ferri carb., gr. xxx.—M. (Pepper.)

Div. in pil. No. xxx.
Sig.—One pill every three hours.

URÆMIA.

Definition.—The name applied to a group of symptoms resulting from the retention of toxic materials in the blood which should have been eliminated by the kidneys.

Symptoms.—It may develop slowly or abruptly, and may manifest any of the following phenomena: Headache, vertigo, delirium, epileptiform convulsions, coma, sudden blind-

ness (unassociated with any retinal change), and transient paralysis from congestion or œdema of the brain or cord.

Pulmonary Symptoms.—Dyspnœa, (uræmic asthma), Cheyne-Stokes breathing.

Abdominal Symptoms.—Hiccough, obstinate vomiting, and purging.

General Symptoms.—The skin is dry; the breath has a urinous odor; the urine is scanty and deficient in urea. The pulse is slow and full, and the temperature subnormal; but during convulsions the temperature may rise and the pulse become rapid and feeble.

Diagnosis.—The various manifestations may be recognized as uræmic by the history, the temperature, the odor of the breath, the high arterial tension, the accentuated second sound of the heart, the presence of casts and albumin in the urine, and by the absence of any other cause.

Prognosis.—Grave, but always guarded, for recovery is possible after the most serious manifestations.

Treatment.—The poison must be eliminated promptly. Croton oil (1 to 2 drops in a little glycerin) or elaterium (gr. $\frac{1}{4}$) should be given to promote catharsis. Free diaphoresis may be secured by hot-air or vapor baths, and the subcutaneous injection of pilocarpine (gr. $\frac{1}{4}$ to $\frac{1}{3}$). When the patient is not too weak venesection is of great service. The subcutaneous administration of a sterilized normal salt solution is highly recommended. Convulsions may be controlled by inhalations of chloroform, and the exhibition by the rectum of chloral hydrate (gr. xxx to xl). Some advise the use of morphine, but in the convulsions of chronic interstitial nephritis it must be used with extreme caution.

ACUTE NEPHRITIS.

(Acute Bright's Disease, Acute Tubular Nephritis, Acute Desquamative Nephritis, Acute Parenchymatous Nephritis, Acute Catarrhal Nephritis.)

Definition.—An acute inflammatory process involving more or less the whole kidney, but especially affecting the epithelium of the tubules and glomeruli.

Etiology.—(1) Exposure to cold and wet. (2) The specific fevers, especially scarlet fever. (3) Poisons which are eliminated through the kidneys, as cantharides, turpentine, etc. (4) Pregnancy.

Pathology.—The kidney is swollen and the capsule non-adherent. At first the organ is bright red in color; it soon, however, becomes pale and mottled in appearance, although the Malpighian tufts still retain their deep red tint.

Histology.—The epithelium of the tubules and glomeruli is the seat of cloudy swelling and, later, of fatty degeneration. Desquamated epithelium, blood-corpuscles, and an albuminous exudate block up the tubules. The capillaries are dilated, their walls degenerated, and bloody extravasations are not infrequently seen. The interstitial tissue is more or less infiltrated with leucocytes.

Symptoms.—Moderate fever and its associated symptoms; dull lumbar pain; nausea and vomiting; dropsy, beginning in the face and becoming general; rapid anæmia. Uræmic symptoms may develop at any time.

The Urine.—Scanty and at times suppressed. It is smoky in appearance, of high specific gravity, rich in albumin, and throws a heavy sediment, which contains hyaline, blood, and epithelial casts, and free blood and epithelial cells.

Diagnosis.—As the general symptoms are often slight, the diagnosis must rest on the examination of the urine. The history, and the absence in the urine of wide, highly fatty casts, will serve to distinguish *acute nephritis* from an *acute exacerbation of chronic parenchymatous nephritis.*

Prognosis.—Guardedly favorable. It may kill by exhaustion, uræmia, or dropsy. It may become chronic.

Treatment.—Absolute rest in bed until albumin has disappeared from the urine. Milk is the best food; but buttermilk, gruels, and light broths are admissible. The free use of water should be encouraged. Dry or wet cups, or hot fomentations should be applied to the loins. To secure vicarious action of the skin vapor baths or small doses of pilocarpine (gr. $\frac{1}{8}$ to $\frac{1}{16}$) may be employed. Concentrated saline draughts, made of Rochelle or Epsom salts, may be given to secure watery discharges from the bowels. Compound jalap powder

(gr. xx), or elaterium (gr. $\frac{1}{8}$) may be substituted for the saline. Stimulating diuretics should be avoided, and diuresis encouraged by alkaline waters and infusion of digitalis. Uræmia will call for its appropriate treatment.

Severe cases in pregnancy will require the induction of abortion or premature labor.

Marked effusions into the serous cavities will sometimes demand aspiration. Convalescence should be protracted, and the resulting anæmia will call for some preparation of iron, such as Basham's mixture.

CHRONIC PARENCHYMATOUS NEPHRITIS.

(Chronic Catarrhal Nephritis, Large White Kidney.)

Etiology.—(1) It may result from acute nephritis. (2) It may be chronic from the beginning. Male sex, adult life, frequent exposure to cold and wet, alcoholism, congestion from heart disease, and syphilis are predisposing factors.

Pathology.—In the first stage the kidney is large and pale-yellow in color; the pallor depends on anæmia and fatty degeneration; the tubes are filled with fatty epithelium and casts; there is always some overgrowth of the interstitial connective tissue.

In the second stage the organ is small, pale in color, its surface rough, and its capsule somewhat adherent. The reduced size depends on destruction of the renal epithelium and the contraction of the overgrown connective tissue.

Symptoms. — As it usually begins as a chronic affection, the following symptoms slowly manifest themselves: Progressive loss of flesh and strength; marked anæmia; gastro-intestinal disturbances; dropsy, often first noted in the face on rising in the morning; increased arterial tension; some hypertrophy of the left ventricle, so that the second sound at the aortic cartilage is accentuated. Uræmic symptoms may develop at any time.

The Urine.—Usually diminished, although it is frequently normal in color and in appearance. It is highly albuminous, and throws down an abundant sediment, which contains hyaline, fatty, and granular casts, and fatty epithelial cells.

Complications.—These are numerous and often suggest the diagnosis. The most common are uræmia, extensive dropsy into the tissues or serous cavities, latent inflammations of the serous membranes, valvular heart disease, albuminuric retinitis, apoplexy, and acute exacerbations.

Prognosis.—Unfavorable. In the early stages recovery sometimes results. The duration is from a few months to several years.

Treatment.—The treatment is largely dietetic and hygienic. Residence in a dry, warm, and equable climate may prolong life or effect a cure. Rest is an essential element in the treatment. The underclothing should be woollen or silk. The diet should be non-nitrogenous, and in severe cases an absolute milk diet may be of extreme value. The bowels should be kept active by natural mineral waters or saline laxatives. When the urine is scanty, digitalis, caffeine, or strontium lactate (gr. xv–xxx) may prove efficient. Basham's mixture may be employed as a chalybeate and a diuretic.

In excessive dropsy promote catharsis by Epsom salts in concentrated solution, or by compound jalap powder; and promote diaphoresis by the hot-air bath, or by pilocarpine.

Niemeyer's pill (page 90) or the following combination is often very efficient in troublesome dropsy:—

℞ Spartein. sulph., gr. vj;
Caffein. citrat., gr. xxx;
Lithii benzoat., ʒj.—M.

Ft. chart. No. xii.
Sig.—One powder every three hours.

Acute exacerbations should be treated as primary attacks of acute nephritis.

CHRONIC INTERSTITIAL NEPHRITIS.

(Red Granular Kidney, Contracted Kidney, Gouty Kidney.)

Definition.—A chronic inflammatory condition of the kidney characterized by a reduction in its size, due to an overgrowth and subsequent contraction of its connective-tissue elements, and invariably associated with general arterial sclerosis and cardiac hypertrophy.

Etiology.—It may be secondary to parenchymatous nephritis, or result from the passive congestion of chronic heart disease; but generally it arises as a primary condition, and results from the causes which predispose to sclerosis in other organs, viz., middle life, male sex, syphilis, the gouty diathesis, chronic alcoholism, and chronic mineral poisoning, as from lead.

Pathology.—The kidneys are small, and red in color. The surface is granular, and the capsule adherent. The organ is firm, cuts with difficulty, and on section often reveals small cysts or calcareous deposits. The cortical substance is greatly reduced in thickness. Microscopic examination shows an overgrowth of connective tissue which has contracted, narrowed the lumen of the tubules, and interfered with the nutrition of the epithelium, and as a result the latter may show fatty degeneration with desquamation. The arteries throughout the body reveal fatty degeneration of the media and an overgrowth of connective tissue in the intima (arteriosclerosis), and from the resistance thus offered hypertrophy of the heart has resulted.

Symptoms.—A slow loss of flesh and strength with progressive anæmia. Gastric disturbances are very common. The arteries are rigid, and the pulse is of high tension, so that the second sound of the heart is accentuated at the aortic cartilage.

Palpitation of the heart is often noted. Dyspnœa is a prominent symptom, and may result from heart-weakness, uræmia, or œdema of the lungs. Headache, vertigo, and insomnia often result from disturbed circulation, and dimness of vision from albuminuric retinitis.

Dropsy is often absent, or is slight and appears late in the disease.

The urine: Increased in quantity, pale in color, and of low specific gravity (1010–1005), and contains but a trace of albumin and a few narrow hyaline casts.

Complications. — Albuminuric retinitis, valvular heart disease, apoplexy resulting from the weakened arteries and large heart, uræmia, latent inflammations of serous membranes, pneumonia, and bronchitis.

Diagnosis.—The arterial changes, casts in the urine, uræmic symptoms, and the absence of poikilocytosis will serve to distinguish chronic nephritis from *pernicious anæmia.*

Chronic parenchymatous nephritis usually occurs earlier in life, lacks much arterial change, produces considerable dropsy, and urine that is rich in albumin and tube-casts.

Prognosis.—It is incurable, but may last many years, and under favorable conditions comparative comfort may be obtained.

Treatment.—The dietetic and hygienic treatment is the same as in chronic parenchymatous nephritis. Frequent bathing with friction of the skin should be encouraged, and the bowels kept regular by alkaline waters.

Absorbents, like the bichloride of mercury and iodide of potassium, are of little value. If the stomach will bear it, iron will be of service. Digitalis, caffeine, and strychnine will be very useful when the heart weakens. Nitroglycerin, in one minim doses, gradually increased, has been recommended for the high arterial tension.

AMYLOID KIDNEY.

(Waxy Kidney, Lardaceous Kidney.)

Etiology.—(1) Prolonged suppuration, particularly in bone disease. (2) Tuberculosis. (3) Syphilis. (4) Malarial cachexia.

Pathology.—The kidney is large and pale, and on section presents a waxy, translucent appearance.

Lugol's solution of iodine strikes a mahogany-red color with the amyloid material.

On microscopic examination, the walls of the bloodvessels, particularly those of the Malpighian tufts, are found thickened, and infiltrated with a homogeneous wax-like material, which turns red when treated with a weak solution of gentian-violet.

Parenchymatous and interstitial changes are always noted. Other organs, especially the liver and spleen, are similarly affected.

Symptoms.—Loss of flesh and strength, with great pallor and moderate dropsy. Uræmic symptoms are uncommon.

The liver and spleen are often much enlarged from the same degeneration.

The Urine.—Usually increased in amount, pale in color, and contains considerable albumin and wide hyaline and granular casts.

Diagnosis.—The history, the enlarged liver and spleen, and the increased amount of urine containing considerable albumin suggest the diagnosis.

Prognosis.—When not advanced, and the cause can be removed, the disease may be arrested. As a rule, the prognosis is decidedly unfavorable.

Treatment.—The primary disease will claim attention. In bone disease, surgical interference may be requisite. In syphilis, iodide of potassium and mercurials will be indicated. In malarial cachexia, iron, quinine, and arsenic should be employed. Tuberculosis will call for its appropriate remedies.

The treatment of the morbid condition is hygienic and dietetic. Alterative tonics, like the iodide of iron, may prove beneficial in some cases.

RENAL CALCULUS.

(Nephrolithiasis, Renal Gravel.)

Definition.—A precipitated urinary concretion found in the kidney.

Etiology.—(1) Male sex. (2) Heredity. (3) Mal-assimilation. (4) Inflammation of the pelvis of the kidney. Doubtless mucus or desquamated epithelium forms the nucleus upon which the stone is built.

Varieties.—(1) Uric acid. This may be passed as sand, or form large reddish-brown stones (2) Oxalate of lime. This forms a very hard, dark, and uneven stone (mulberry calculus). (3) Phosphates. These are composed of phosphate of lime, and ammonio-magnesium phosphate, and are soft, mortar-like in appearance, and are often deposited on other calculi. (4) Xanthine and cystine are rare concretions.

Events.—(1) A stone may remain latent indefinitely. (2) It may pass out, with or without the symptoms of colic. (3) It

excites pyelitis, and sometimes abscess of the kidney. (4) It may obstruct the ureter and produce hydro-nephrosis or pyo-nephrosis. (5) It may excite perinephritis, and may perforate in other organs.

Symptoms of Renal Colic.—Sudden onset, with sharp pain, starting in the back and radiating down the ureter into the penis, testicle, or thigh. There may be retraction of the testicle on the affected side.

The symptoms of intense pain are often present, viz: pallor, cold sweats, weak pulse, and reflex vomiting.

The urine subsequently passed may contain the stone; or, as a result of irritation, pus, blood and desquamated pelvic epithelium. An attack may last from a few moments to several hours.

Diagnosis. *Biliary and Renal Colic.*—In the former the pain runs from the right hypochondriac region to the right shoulder; there is often jaundice, and the urine is negative, while the stools may contain the stone.

Prognosis.—In view of the complications the prognosis must be guarded.

Treatment. *The Attack.*—Morphine and atropine should be employed hypodermically, and warm poultices applied to the loins. The free use of water should be encouraged. In severe cases chloroform or ether may be inhaled in sufficient quantity to obtund the sensibility of the patient.

The Interval.—When symptoms persist, regulate the diet, and put the patient under good hygienic conditions. When the reaction of the urine indicates an acid stone, the salts of lithium or the vegetable salts of potash may be employed in large doses, over long periods. A drachm of the citrate of potassium or five to ten grains of the carbonate of lithium may be given, well diluted, several times a day. The natural mineral waters are of some value. The Buffalo lithia water may be employed for this purpose, and its palatableness and efficiency may be increased by the addition of a teaspoonful of some effervescing preparation of lithium to each potation.

When an alkaline stone is indicated, benzoic acid or boric acid may be employed in a similar manner.

In severe and persistent cases the stone may be excised

(nephro-lithotomy); and if the operation should reveal a badly-damaged kidney, its removal (nephrectomy) would be indicated.

PYELITIS.

(Pyelonephritis, Pyonephrosis.)

Definition.—Inflammation of the pelvis of the kidney.

Etiology.—(1) It may result from stone in the pelvis of the kidney (calculous pyelitis). (2) It may be secondary to urethritis or cystitis extending upwards through the ureters. (3) It may follow pregnancy or the specific fevers. (4) Morbid growths, such as tubercle or cancer. (5) Toxic doses of the stimulating diuretics (copaiba, cantharides, etc.). (6) It is rarely idiopathic from exposure to cold and wet.

Pathology.—The mucous membrane is swollen, injected, and covered with a tenacious secretion composed of mucus, pus, and desquamated epithelium. Severe cases may lead to dilatation of the pelvis, Bright's disease, or suppurative nephritis.

Symptoms.—Moderate fever and its associated phenomena. In suppurative nephritis the fever may be irregular and associated with hectic or typhoid symptoms. There is pain and sometimes tenderness over the kidneys. The urine is turbid, acid in reaction, and on standing throws down a sediment containing considerable mucus, pus-corpuscles, pelvic epithelium, and blood-corpuscles. The pus and blood render the urine slightly albuminous.

Diagnosis.—The absence of much albumin, of tube-casts, and dropsy exclude *nephritis*.

Cystitis may be detected by the absence of lumbar pains and of acid urine, and by the presence of frequent and painful micturition and alkaline urine containing vesical epithelium.

Perinephritic abscess is also associated with lumbar pain and hectic fever; but in addition there is often œdema over the lumbar region, and the urine may be normal.

Sharp pain over the kidney, increased by jarring movements, and reflected down the ureters, and the presence of much blood in the urine point to *calculous pyelitis*.

Tuberculous pyelitis may be recognized by the history, by the presence of tubercle in other organs, and by tubercle bacilli in the urine.

Pyelitis secondary to cystitis is recognized by the history.

Prognosis.—Depends on the cause. Mild forms resulting from pregnancy, specific fevers, or exposure to cold, usually recover in a few weeks. The tuberculous and suppurative varieties are unfavorable.

Treatment.—Depends on the cause. Calculous pyelitis will require the treatment indicated for renal calculus. In simple pyelitis keep the patient at rest, restrict the diet to light food, preferably to milk, apply warm poultices locally, use alkaline diluents and some sedative mixture, as the following :—

℞ Potass. bromid.,
Sodii bicarb., āā gr. clx ;
Ext. belladonnæ, gr. iv;
Ext. buchu, ʒj ;
Syr. sarsp. comp., q. s. ad fʒiv.—M. (Pepper.)
Sig.—Tablespoonful three times a day.

In pyelitis following cystitis, treat the latter locally, and use stimulating diuretics, like eucalyptus, sandalwood, and copaiba.

HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis of the kidney, with the accumulation of a watery fluid, resulting from obstruction.

Etiology.—(1) Congenital stricture of the ureter. (2) Impaction of a calculus in the ureter. (3) Abdominal tumors compressing the ureter. (4) Tumors growing within the urinary passages. (5) An inflammatory stricture of the ureter or urethra.

Pathology.—The pelvis reveals all grades of distention. In extreme cases it may contain several quarts of fluid, which is at first urinous, but later thin and watery. There is more or less atrophy of the renal tissue.

Symptoms.—Slight distention yields no symptoms. In other cases a tumor slowly develops in the region of the affected kidney. On palpation it is elastic, and perhaps

fluctuating; on percussion, dull; and on aspiration it yields a clear fluid, which usually contains urea and uric acid.

Diagnosis.—This will be based on the history, the exclusion of other abdominal enlargements, and the chemical analysis of the fluid obtained by aspiration.

Prognosis.—Usually unfavorable. When it is unilateral, and the other kidney secretes a normal amount of urine, containing a normal amount of urea, the prognosis is guardedly favorable.

Treatment.—When the distention is moderate the treatment is expectant. When the sac is large, aspirate; and if re-accumulation is rapid, establish a renal fistula or remove the organ.

FLOATING KIDNEY.

(Movable Kidney.)

Definition.—A distinctly mobile condition of the kidney, dependent upon a relaxation of the tissues which surround it.

Etiology.—(1) Female sex. (2) Middle life. (3) Rapid emaciation leading to the absorption of the perinephritic fat. (4) A congenital relaxed condition of the perinephritic tissues. (5) Muscular exertion. (6) Repeated pregnancies.

Symptoms.—The right kidney is the one usually affected, probably from its relation to the liver, which moves during the respiratory acts. The kidney may be found in any part of the abdomen, as a movable tumor, reniform in shape, somewhat tender to the touch, and rarely imparting the pulsation of the renal artery.

There may be no subjective symptoms, but a sense of uneasiness and attacks of neuralgic pain are often noted. At times the kidney may become swollen and very tender, probably from twisting of the renal vessels inducing engorgement of the organ. Emotional disturbances are often excited by the condition.

Diagnosis.—The reniform shape of the tumor, its free mobility, its stationary size, the lessened resistance on percussion over the renal region of the affected side, and the absence of cachexia will serve to diagnose a floating kidney from other abdominal tumors.

Treatment.—In many cases, a regulated diet, the avoidance of undue exertion, and the use of a broad binder applied firmly to the abdomen will be the only treatment required. When the symptoms persist the kidney may be stitched in its normal place (nephrorrhaphy); and if this treatment fails the offending organ may be removed (nephrectomy).

TUBERCULOSIS OF THE KIDNEY.

Etiology.—The etiology of renal tuberculosis is that of tuberculosis in general. Males are more frequently attacked than females. The majority of cases are encountered between the ages of twenty and forty years.

Pathology.—Two forms of renal tuberculosis have been recognized—the miliary and the caseous. The former is nearly always bilateral, is an acute process, and is generally unmistakably secondary to tuberculosis elsewhere in the body. The caseous variety runs a chronic course; it usually begins as a unilateral affection, although the other organ is commonly ultimately involved, and a primary focus may or may not be apparent in some other structure.

Symptoms.—Pain in the lumbar region, usually dull, but sometimes sharp, like that of renal colic; tenderness on pressure; slight, irregular fever, and more or less cachexia. The urine is usually acid in reaction, and may contain pus, blood, albumin, tubercle bacilli, cheesy particles, and *débris*. Tube-casts are rarely found. In many cases enlargement of the affected organ can be detected by bimanual palpation.

Diagnosis. *Calculous Pyelitis.*—In this condition pain is usually more severe, and more apt to be affected by movement. Hæmaturia is more profuse, and is often excited by exertion. Cachexia is not so marked, and there are no tubercle bacilli in the urine.

Prognosis.—Always grave. Without intervention the duration is from a few months to three years.

Treatment.—When the renal disease appears to be primary and the patient's strength will permit, nephrectomy should be recommended. The mortality in operative cases has been about 28 per cent. In other cases the treatment must of necessity be palliative.

DISEASES OF THE BLOOD AND THE DUCTLESS GLANDS.

NORMAL BLOOD.

In health the blood amounts to about one-thirteenth of the body-weight. Normally there are approximately 5,000,000 red blood-corpuscles in the cubic millimetre. This number is temporarily diminished during menstruation, gestation, lactation, and fatigue, and after the ingestion of much fluid. Fasting and profuse sweating increase the number of red cells by concentrating the blood. In the first few days of life the number per cubic millimetre may be 7,000,000 to 8,000,000. In high altitudes the number is also increased. There are from 5,000 to 10,000 white cells in the cubic millimetre, the ratio of white to red cells being about 1 to 500. The number of blood plates is from 200,000 to 300,000.

EXAMINATION OF THE BLOOD.

A clinical study of the blood has for its object the determination of the percentage of hæmoglobin, the specific gravity, the alkalinity, the number, form, and relative proportion of the various corpuscles, and the detection of free pigment, bacteria, and animal parasites.

Estimation of Hæmoglobin.—The percentage of hæmoglobin may be determined by either Fleischl's or Gowers' apparatus, although the former is preferable.

Gowers' hæmoglobinometer consists of (1) a small sealed tube containing coloring matter representing the color of normal blood diluted with 100 parts of water; (2) an empty tube of the same size, graduated up to 120 per cent.; (3) a small bottle with a pipette stopper, for distilled water; (4) a capillary

pipette for measuring 20 c.mm. of blood; and (5) a small lancet. To obtain a specimen of blood the tip of the finger or the lobe of the ear, after being thoroughly cleansed, is deeply pricked with the lancet, so that the blood flows freely without squeezing; 20 c.mm. of blood are then drawn into the capillary pipette, and are immediately blown into the graduated tube, in which have been previously placed a few drops of distilled water to prevent coagulation. After shaking the mixture to secure diffusion of the blood, more distilled water is cautiously added, with occasional shaking, until the tint in the sealed tube is reached. The height of the column of the fluid in the graduated tube will indicate the percentage of hæmoglobin.

Fleischl's instrument consists of a metal stand with a circular aperture in the centre, under which is placed a reflector made of plaster-of-Paris. The aperture is fitted with a small cell having a glass bottom, and divided into two equal compartments. A graduated wedge of colored glass is employed as a standard, the 100 on the scale being intended to represent the percentage of hæmoglobin in normal blood. This wedge of glass is so arranged that when moved under the stand, one compartment of the cell will receive white light from the reflector, and the other, red light from the tinted glass. A small capillary tube is held over a drop of blood until filled, and is then washed in one of the compartments of the cell, in which has been previously placed some distilled water. Both compartments are then equally filled with water, and the wedge of glass is moved by means of a thumb-screw until the tints in the two chambers are exactly the same, when the percentage of hæmoglobin may be read off.

In the examination it is necessary to use artificial light. The 100 mark on the scale, which is intended to represent the percentage of hæmoglobin in normal blood, is too high for the average person, 85 or 90 per cent. rarely being exceeded.

The Specific Gravity of the Blood.—The specific gravity of the blood in health varies from 1050 to 1070. In grave anæmia it is often considerably diminished. Hammerschlag's method consists in expelling a drop of blood into a mixture of chloroform and benzol, one or the other of these

substances being subsequently added until the drop neither rises nor falls. The specific gravity of the mixture may then be ascertained in the usual way. Lloyd Jones employs mixtures of glycerine and water of different densities, and notes the specific gravity of the mixture in which the blood-drop remains stationary.

Alkalinity of the Blood.—The alkalinity of the blood may be determined by titrating with a standard solution of acetic acid until a change of color is produced when a drop is placed on a plaster-of-Paris plate impregnated with neutral litmus.

Enumeration of Corpuscles.—The best instrument for estimating the number of corpuscles is the *hæmocytometer* of Thoma-Zeiss. This consists of a glass slide, in the centre of which is a cell $\frac{1}{10}$ mm. in depth. The floor of the cell is divided into squares, the sides of which are $\frac{1}{20}$ mm. Twenty-five small squares constitute a large square, which is indicated by heavy lines. The blood is mixed in a *mélangeur*—that is, a capillary tube one extremity of which is blown into a bulb having a capacity of 100 c.mm. The mélangeur is marked at 0.5, 1 c.mm. and 101 c.mm. A drop of blood issuing from a prick of the finger or lobe of the ear is drawn cautiously into the tube to the 1 c.mm. mark. The point is quickly wiped and immersed in the diluting fluid ($2\frac{1}{2}$ per cent. solution of potassium bichromate) which is drawn up to the 101 c.mm. mark. The instrument is now shaken to secure diffusion of the blood. The diluting fluid remaining in the stem of the mélangeur is now blown out, and a drop of the mixture placed in the blood-counting cell. The drop in the cell should be free from bubbles, and the cover-glass so adjusted that Newton's rings appear at the margin of the drop. Before counting, a few minutes should be allowed for the corpuscles to settle to the bottom of the cell. The number of corpuscles is then counted in sixteen large squares (400 small squares), the average number in each small square being determined by dividing the whole sum by 400. This number is then multiplied by 400,000,—100 for the dilution, and $20 \times 20 \times 10$ for the cube of the cell.

After using, the mélangeur should be carefully washed in water, alcohol, and ether.

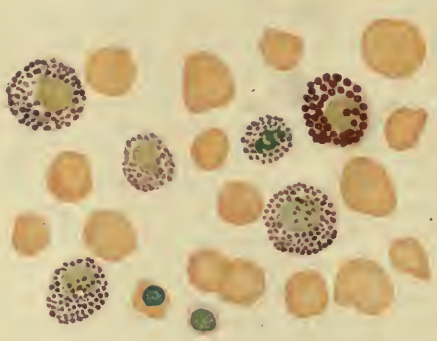
The Study of the White Blood-corpuscles.—In normal blood, five varieties of white blood-corpuscles may be observed:

1. *Lymphocytes*, or small mononuclear forms, about the size of red blood-corpuscles, with large deeply staining nuclei, and a narrow margin of non-granular protoplasm. They have their origin in the lymph-glands, and constitute about 20 to 30 per cent. of all the leucocytes.

2. *Large mononuclear cells*, three or four times the size of the red blood-corpuscles, with oval nuclei, surrounded by non-granular protoplasm.

3. *Transitional forms*, differing from the large mononuclear cells only in having nuclei with indentations. The large

FIG. 7.



Blood in lieno-medullary leukæmia, showing several mononuclear neutrophiles (myelocytes), one polymorphonuclear neutrophile, and an eosinophile; a nucleated red corpuscle and a lymphocyte are seen in the lower part of the illustration. Stained with Ehrlich's triple mixture. (From Stengel's *Text-Book of Pathology.*)

mononuclear forms constitute from 6 to 8 per cent. of the whole number of leucocytes.

4. *Polynuclear Forms, or Leucocytes with Polymorphous Nuclei.*—These are somewhat smaller than the large mononuclear forms, and contain deeply-staining nuclei which are very

variable in shape. The protoplasm contains abundant neutrophilic granules (*neutrophiles*)—that is, granules which have an affinity for a combination of acid and basic stains. The polynuclear forms, or neutrophiles, are apparently derived from the spleen and bone-marrow, and constitute from 70 to 80 per cent. of all forms.

5. *Eosinophiles.*—These resemble in general appearance the polynuclear forms, but the granules are larger, more highly refractive, and have a special affinity for acid stains, particularly eosin. They have their origin in the bone-marrow, and constitute from 2 to 4 per cent. of all forms.

In addition to the normal elements above mentioned, certain other forms may be observed in the blood of disease. Thus in leukæmia large mononuclear forms (*myelocytes*) are often met with, the protoplasm of which is studded with neutrophilic granules. In the same disease, the blood occasionally contains cells resembling those normally found in connective tissue (*Mastzellen*). They are peculiar in having granules which have an affinity only for basic stains.

With the aid of a one-twelfth inch oil-immersion lens, large and small leucocytes can be readily distinguished in preparations of fresh blood, but to study satisfactorily the various forms it is necessary to dry and then stain the specimen.

The Drying and Staining of Blood.—A small drop of blood, secured by pricking the finger, is spread into a film by being pressed between two perfectly clean cover-glasses, which are then drawn apart and exposed to the air until dry. The cover-glasses should be handled with forceps, since the moisture of the fingers distorts the corpuscles. The preparation is first "fixed" by heating on a copper bar for several hours at a temperature of 110° to 120° C., or by immersing for from fifteen minutes to half an hour in a mixture of equal parts of absolute alcohol and ether. A convenient method of staining is the one suggested by Stengel. The fixed preparation is immersed for a couple of minutes in a 1 per cent. solution of eosin in 60 per cent. alcohol, to which has been added an equal quantity of water at the time of staining. The cover-glass is then washed in water and counter-stained in

Delafield's hæmatoxylin for a minute, and finally washed, dried, and mounted. The eosinophile granules are dark red, the red corpuscles lighter red, and the nuclei of the white blood-corpuscles almost black. Thayer recommends the following solution:

Solution:

Saturated aqueous solution of acid fuchsin	2 parts.
Water	3 "
Saturated aqueous solution of orange-green	6.25 "
Saturated aqueous solution of methyl-green	6 "

To which is added drop by drop while the solution is shaken:

Water	15 parts.
Alcohol	10 "
Glycerin	5 "

The fixed specimen is stained in this solution for from three to four minutes, washed in water, dried in the air, and mounted in balsam. The nuclei of white blood-corpuscles appear green, the eosinophile granules dark red, the neutrophile granules violet, the red blood-corpuscles orange, and the nuclei of any existing nucleated red blood-corpuscles dark green.

PLETHORA.

An increase in the whole quantity of blood. It is very doubtful whether such a condition can be more than transitory.

HYDRÆMIA.

An excess of water in the blood. As a loss of corpuscular elements is generally replaced by the addition of water extracted from the tissues, most anæmias are associated with hydræmia. The condition is more marked in general dropsy. Temporary hydræmia is produced by the excessive ingestion of fluids.

ANHYDRÆMIA.

A deficiency of fluid in the blood. It is observed in starvation, immediately after hemorrhage, and after copious discharges, as in cholera.

MELANÆMIA.

Melanæmia, or the presence of free pigment in the blood, usually results from chronic malarial infection. It is occasionally associated with melanosarcoma and Addison's disease.

POLYCYTHÆMIA.

Polycythæmia, or an increase in the number of red cells, is an apparent condition in blood taken from cyanosed parts. It is observed temporarily in the new-born, in recovery from certain anæmias, after transfusion of blood, and in blood concentrated by excessive discharges. Marked polycythæmia is sometimes produced by residence in high altitudes and by certain poisons, such as phosphorus and carbon monoxide.

MICROCYTOSIS AND MACROCYTOSIS.

Microcytosis and macrocytosis are conditions in which the red cells are respectively diminished and increased in size. They may occur in any form of severe anæmia, but they are especially marked in pernicious anæmia.

POIKILOCYTOSIS.

Poikilocytosis, a condition in which the red cells are irregular in shape, is common in grave anæmias, especially pernicious anæmia.

NUCLEATED RED CELLS.

Nucleated red cells are divided into three forms—normoblasts, macroblasts, and microblasts. The first resemble in size and color a normal red cell, the second are larger, and the third smaller. Nucleated red cells are not found normally in the circulating blood; they are present, however, in grave forms of anæmia.

LEUCOCYTOSIS.

Leucocytosis, or hyperleucocytosis, is an increase in the number of white blood-corpuscles. It occurs physiologically in the new-born, during digestion, in pregnancy, after parturition, and after massage, exercise, or cold bathing. In physiological leucocytosis the relative proportions of the different forms of white cells to each other are not materially changed.

Pathological leucocytosis is observed in the following conditions:

1. Inflammation. There is an absolute increase in the polynuclear forms.
2. Infectious diseases. Most infections excite leucocytosis, but the condition is often wanting in typhoid fever, malaria, measles, influenza, and tuberculosis. In any infection in which the toxæmia is intense, or the resistance of the individual is slight, leucocytosis may be wanting.
3. Malignant disease.
4. Hemorrhage.
5. Toxæmia. Under this head are included jaundice, uræmia, ptomaine-poisoning, and gout. Many chemical substances—coal-gas, phosphorus, quinine, etc.—also produce it.

LEUCOPÆNIA.

Leucopænia is a diminution in the number of white cells. It is observed in certain infections, particularly those which do not produce leucocytosis, in inanition, and in pernicious anæmia.

LIPÆMIA.

Lipæmia, the presence in the blood of minute fat-globules, may be noted in health. Abnormal quantities of fat are observed in diabetes, alcoholism, and in conditions associated with deficient oxidation, such as phthisis and emphysema.

BLOOD PARASITES.

The following parasites have been detected in the blood: Filaria sanguinis hominis, plasmodium malariæ, spirochætc of relapsing fever; bacilli of anthrax, glanders, typhoid fever, tuberculosis, tetanus, and influenza; diplococcus pneumoniæ, streptococcus, staphylococcus, gonococcus, and bacillus coli communis.

OLIGOCHROMÆMIA.

Oligochromæmia, or deficiency of hæmoglobin, is usually proportionate to the reduction in the number of red cells, but there are two exceptions, namely, in chlorosis, in which disease the red cells may be reduced only 20 or 30 per cent., while the hæmoglobin may be reduced 50 or 60 per cent., and in pernicious anæmia, in which disease the blood-count is very low, while the corpuscles are relatively rich in hæmoglobin.

The *color index* represents the relation between the number of cells and the quantity of hæmoglobin. In a patient having 2,500,000 red cells per cubic millimetre (50 per cent.), and 40 per cent. of hæmoglobin the color index would be $\frac{40}{50} = 0.8$.

OLIGOCYTHÆMIA.

Oligocythæmia, a diminution in the number of red cells, occurs in all forms of anæmia, but it is especially marked in pernicious anæmia and in advanced malignant disease, where the number may fall below a million per cubic millimetre.

ANÆMIA.

Anæmia is a condition in which the blood is deficient in quantity or in one or more of its constituents.

Varieties.—(1) Symptomatic or secondary anæmia. (2) Essential or primary anæmia.

Symptoms.—All forms of anæmia have the following symptoms in common: Pallor of skin and mucous membranes, loss of strength, and, in severe cases, febrile paroxysms and ecchymoses.

Circulation.—A full, soft, and rapid pulse, unnatural pulsation of the cervical vessels, palpitation of the heart, hæmic murmurs, and slight dropsy, beginning in the feet.

Respiration.—Hurried breathing.

Digestion.—Dyspepsia.

Nervous System.—Headache, vertigo, disturbed sleep, neuralgic pains, and tendency to syncope.

Symptomatic Anæmia.

Etiology.—Symptomatic or secondary anæmia usually results from one of three causes: (1) Insufficient nutriment entering the circulation (inadequate food, bad air, chronic gastritis, cancer of the pylorus, etc.). (2) Excessive demands upon the blood-making organs (overwork, hemorrhage, chronic diarrhœa, etc.). (3) Action of toxic agents (lead, malaria, syphilis, uræmia, etc.).

Symptoms.—In addition to the ordinary phenomena of anæmia the blood-count reveals a decrease in the number of red cells and a proportionate deficiency in the percentage of hæmoglobin. The number of polynuclear leucocytes is often increased. In severe form, microcytes, macrocytes, and poikilocytes are present, and rarely nucleated red cells.

Prognosis.—This depends on the cause.

Treatment.—This includes the removal of the cause, when possible; the adoption of hygienic measures, and the use of iron, arsenic, and general tonics.

PERNICIOUS ANÆMIA.

Definition.—A grave form of anæmia, characterized by a great deficiency in the number of red cells and unassociated with any definite causal lesion.

Etiology.—Forms of anæmia clinically identical with pernicious anæmia may be dependent upon the presence of certain intestinal parasites (bothriocephalus and anchylostoma), may follow parturition, or may result from advanced atrophy of the stomach. In many cases no adequate cause is apparent. The disease usually appears about middle life, and

is more frequent in males. According to one theory, it results from increased hæmolysis excited by poisons absorbed from the intestinal canal; according to another, it is due to defective hæmogenesis.

Pathology.—The skin has a lemon-yellow hue, the subcutaneous fat is often well preserved, and the muscles are unusually red. The organs are the seat of fatty degeneration. The gastric tubules are sometimes atrophied. The liver contains an excess of iron, the pigment being distributed especially in the outer and middle zones of the lobules. The bone-marrow is dark red, soft, and contains a large number of nucleated red cells, especially maeroblasts. In many cases there is found advanced selerosis in the posterior columns of the spinal cord.

Symptoms.—Intense anæmia, with its usual symptoms; a lemon-yellow tint to the skin; progressive weakness without much emaciation; moderate, irregular fever; marked gastric disturbances; and sometimes dark-colored urine from the presence of urobilin.

The Blood.—The drop is pale and watery. Coagulation is slow. There is a great reduction in the number of red cells, often to 1,000,000 or less; the hæmoglobin is also reduced, but not proportionately; the red cells are irregular in size and shape (microcytes, macrocytes, and poikilocytes), and there are many nucleated red cells present, especially the large forms (macroblasts). The number of white cells is not increased.

Diagnosis.—*Atrophy of the gastric tubules* may be recognized by analysis of the gastric juice, and the presence of intestinal parasites by examination of the feces. In obscure *cancer* the anæmia may be as intense, but there is usually marked leucocytosis.

Prognosis.—Pernicious anæmia usually ends fatally within one or two years. Recovery is rare, but periods of improvement are of frequent occurrence.

Treatment.—Apart from hygienic measures, arsenic is the only reliable remedy. Two minims of Fowler's solution may be given after each meal and gradually increased, so that at the end of three weeks the patient is taking 20

minims thrice daily. Œdema of the eyelids and gastric disturbance are indications of intolerance, and call for the temporary suspension of the drug. Raw red bone-marrow (one or two ounces daily), with equal parts of glycerin, is a useful adjuvant. Inhalations of oxygen and subcutaneous injections of normal salt solution may give temporary relief in grave cases.

LEUKOCYTHÆMIA.

(**Leukæmia.**)

Definition.—A disease characterized by great excess of the white corpuscles, with lesions of the spleen, lymphatic glands, or bone-marrow.

Etiology.—The causes are obscure. Male sex, middle life, heredity, malaria, bad hygienic conditions, and repeated hemorrhages are predisposing factors. It is probably an infectious disease.

Varieties.—Spleno-medullary and lymphatic; the first is the more common.

Pathology.—There is extreme emaciation. The heart and large veins are filled with clots of a greenish color and puriform appearance. The spleen is much enlarged from a true hyperplasia. It is of a brownish color, and is often studded with pale gray lymphoid nodules. The liver is often enlarged from the infiltration of leucocytes between the liver cells and from the presence of distinct lymphoid growths. The organs generally may be the seat of leukæmic nodules—*i.e.*, masses of proliferating leucocytes. In the medulla of the long bones the fat is replaced by material resembling pus (pyoid marrow), which histologically is composed of numerous nucleated red cells and white cells, many of the latter being myelocytes. The lymphatic variety is characterized by marked hyperplasia of all the lymphatic glands. In the spleno-medullary form the lymph-glands are often more or less enlarged.

Symptoms.—To the ordinary manifestations of intense anæmia are added the following symptoms: Moderate, irregular fever; hemorrhages from mucous membranes; enlargement of the spleen, liver, and lymphatic glands; and sometimes

dimness of vision from retinal hemorrhage or leukæmic deposits.

The Blood of Spleno-medullary Leukæmia.—Examination reveals a reduction in the number of red cells, and a great excess of white cells (100,000 to 800,000), many of the latter being myelocytes—*i. e.*, large mononuclear forms containing fine neutrophilic granules, and not found in normal blood.

The Blood of Lymphatic Leukæmia.—Examination reveals a reduction in the number of red cells and an excess of white cells, about 90 per cent. of the latter being lymphocytes.

Diagnosis. *Leucocytosis.*—In this condition the excess of leucocytes is chiefly of the polynuclear forms.

Hodgkin's disease resembles lymphatic leukæmia, but in the former there is an absence of leucocytosis.

Prognosis and Treatment.—Recovery rarely follows. Death usually results in from one to three years; acute cases may terminate fatally in the course of a few weeks. The treatment is the same as that of pernicious anæmia.

PSEUDO-LEUKÆMIA.

(Hodgkin's Disease, Lymphatic Anæmia, Malignant Lymphoma.)

Definition.—A disease characterized by hyperplasia of the lymphatic glands and progressive anæmia, without a marked excess of white corpuscles.

Etiology.—The causes are unknown. It is most commonly seen in young adults of the male sex. In some instances it has apparently followed an ordinary adenitis. An infectious origin has been suggested.

Pathology.—There is a marked hyperplasia of the lymphatic glands—cervical, axillary, mediastinal, inguinal, and retroperitoneal, the spleen and bone-marrow often sharing in the process.

Symptoms.—The disease resembles lymphatic leukæmia, but there is an absence of leucocytosis. The glands of the neck are usually first affected; the swellings are painless, freely movable, and only very rarely suppurate.

DIAGNOSIS.—*Tuberculous adenitis* is more apt to affect the submaxillary glands and is often unilateral; moreover, suppuration of the glands is common in tuberculosis.

PROGNOSIS AND TREATMENT.—Hodgkin's disease is almost invariably fatal. The duration is from a few months to three years. The treatment is the same as that of pernicious anæmia.

CHLOROSIS.

(Green Sickness, Primary Anæmia.)

ETIOLOGY.—The predisposing causes are age (puberty), sex (females, rarely males), and bad hygiene (poor food, impure air, overwork, and lack of sunlight). The absorption of ptomaïnes from the bowel has been suggested as the exciting cause.

PATHOLOGY.—In some fatal cases imperfect development of the circulatory system and of the genitalia has been observed.

SYMPTOMS.—Anæmia with its usual manifestations; a very marked reduction in the hæmoglobin without a corresponding reduction in the number of red blood-cells; a greenish tint of the skin; a capricious appetite (pica); constipation; pallor and weakness without loss of flesh; and a tendency to hysterical outbreaks and to menstrual disorders.

COMPLICATIONS.—Gastric ulcer, dilatation of the stomach, gastralgia, amenorrhœa, phthisis, exophthalmic goitre, and thrombosis.

PROGNOSIS.—Appropriate treatment is followed by a speedy recovery, but relapses are common.

TREATMENT.—The duration of the disease is materially shortened by rest and the regulation of the diet. The constipation should be relieved by saline laxatives. The special remedy is iron, which should be given in ascending doses.

℞ Ferri sulphatis ex.,
Potassii carbonatis, āā gr. xl.—M.
Ft. in pil. No. xx.
Sig.—Three pills daily, increased to nine pills daily.

ADDISON'S DISEASE.

DEFINITION.—A constitutional disease, characterized anatomically by a degeneration of the suprarenal capsules, and clinically by pigmentation of the skin, anæmia, and prostration.

ETIOLOGY.—Male sex, middle life, and laborious work are predisposing factors.

PATHOLOGY.—In most instances tuberculosis of the suprarenal capsules is discovered. Other affections, such as tumors and degeneration of the suprarenal capsules, may produce the disease. In a few instances degenerative changes in the abdominal sympathetic ganglia have been the only discoverable lesions.

SYMPTOMS.—Moderate anæmia, with bronzing of the skin and mucous membranes, great weakness, and gastric irritability are its chief manifestations.

PROGNOSIS.—The disease has been considered incurable, death generally resulting in from one to two years; but recently good results have followed the ingestion of suprarenal glands.

TREATMENT.—The general treatment includes rest, a nutritious but easily assimilable diet, and the use of tonics like iron, arsenic, quinine, and strychnine. A glycerine extract of two fresh suprarenal capsules, or an equivalent amount of dried extract, should be taken daily.

EXOPHTHALMIC GOITRE.

(Graves's Disease, Basedow's Disease.)

DEFINITION.—A nervous affection, characterized by protrusion of the eyeballs, enlargement of the thyroid gland, and palpitation.

ETIOLOGY.—Early adult life, female sex, and nervous temperament are the predisposing causes. It sometimes develops suddenly under emotional excitement, such as fright, grief, and anxiety.

PATHOLOGY.—The pathogenesis of Graves's disease is still undetermined. Some attribute the phenomena to a lesion of the medulla, others to a disturbance of the sympa-

thetic nervous system, while modern research indicates that the chief factor is overaction of the thyroid gland (hyperthyroidation). Greenfield found the tubular spaces of the gland proliferated and the colloid matrix replaced by a more mucoid material.

Symptoms. *Cardiac Phenomena.* — Acceleration of the pulse (100–150) and palpitation, both greatly exaggerated by excitement; hypertrophy of the heart from its rapid action; occasionally a soft systolic murmur at the apex.

Ocular Phenomena.—Bilateral protrusion of the eyeballs; Graefe's sign, which consists in a failure of the upper lid to follow the eyeball when the latter is directed downwards; widening of the palpebral angle (Stellwag's sign). Vision is usually unimpaired.

Thyroid Phenomena.—Enlargement of the thyroid is often the last symptom to appear; one or both lobes of the gland may be affected. Inspection reveals enlargement with pulsation; palpation detects a soft swelling and a purring thrill; auscultation may yield a *bruit.*

Nervous Phenomena.—The following are sometimes observed: A tremor of the hands or of the entire body; hypochondriasis; acute mania; or vitiligo and chloasma.

General Phenomena. — Anæmia, failure of health and strength, and slight febrile paroxysms.

Diagnosis.—It should be borne in mind that one of the three important symptoms may be absent throughout the disease. In some cases palpitation and throbbing of the cervical vessels may be the only phenomena.

Goitre may be distinguished from exophthalmic goitre by the absence of cardiac, ocular, and nervous symptoms.

Prognosis.— The disease generally runs a protracted course. Some cases recover entirely; many improve and subsequently relapse; a few die, after a short illness, from heart failure or acute mania.

Treatment.—The general nutrition must be improved by rest, a liberal diet, and the use of such tonics as iron, quinine, and arsenic. The application of mild galvanic currents to the neck is often very useful. When the palpitation is marked, prompt relief often follows absolute rest and the application

of an ice-bag to the præcordia. The most reliable internal remedies are strophanthus, digitalis, belladonna, and ergot. Bromide of potassium is sometimes useful in controlling the nervous symptoms. Thyroid extract is harmful.

Operative treatment is hazardous, though not infrequently followed by excellent results.

MYXŒDEMA.

Definition.—A constitutional affection, characterized by mucoid degeneration of the subcutaneous tissues, atrophy of the thyroid gland, and mental impairment.

Etiology.—The disease is much more frequent in women than in men. It is occasionally hereditary. It usually develops in middle life. The immediate cause is atrophy of the thyroid gland.

A congenital form of myxœdema is observed in cretinism, and an analogous condition (operative myxœdema or cachexia strumipriva) frequently follows total extirpation of the thyroid gland.

Symptoms.—It is manifested by a gradual swelling, particularly marked in the face, supraclavicular regions, and hands. Unlike œdema, the parts do not pit on pressure, but are firm and elastic. The skin is dry and harsh. The hair is dry and brittle. The thyroid gland is atrophied. A peculiar slowness in thought, speech, and movements is a characteristic symptom. The temperature of the body is subnormal. There is impairment of the special senses. Sensory phenomena are common, such as coldness, numbness, and tingling. The urine is often increased in quantity, and occasionally contains albumin, sugar, and tube-casts.

Complications.—Insanity, tuberculosis, exophthalmic goitre, and nephritis.

Diagnosis.—The mental dulness, the extreme dryness of the skin, the absence of pitting on pressure will separate myxœdema from Bright's disease with œdema.

Prognosis.—The disease was formerly considered incurable, but it is now known that marked amelioration or even a cure can be effected by appropriate treatment.

TREATMENT.—Murray was the first to demonstrate the value of thyroid juice in myxœdema. A glycerine extract or a dried extract of the gland may be employed; the latter is very efficient in doses of one grain, gradually increased to five grains, three times a day. Residence in a warm climate is desirable. Warm baths followed by friction and massage are useful.

DISEASES

OF THE

CIRCULATORY SYSTEM.

INSPECTION.

Inspection detects the apex-beat, and determines its position, force, and extent; any abnormal centres of pulsation; and any unnatural prominence over the præcordial region.

The Apex-beat.

The normal position of the apex-beat is in the fifth intercostal space, about an inch within the mammary line (a line drawn from the middle of the clavicle parallel with the sternum). The beat is usually detected by inspection or palpation, but when these methods fail it may be localized by auscultation, the point in the region of the apex where the first sound is heard with maximum intensity corresponding to the beat.

The Effect of Respiration and Position on the Apex-beat.—The location and force of the apex-beat are modified by the posture of the patient and the stage of the respiratory act. In the recumbent position the apex-beat may be elevated an inch or more, and when the body is inclined to the left, the heart being a more or less movable organ, the beat may be detected in the mammary line, or even some distance to its outer side.

During forced inspiration the beat may become imperceptible, or if such is not the case it may be found some distance below its usual place, on account of the upward

movement of the ribs in the inspiratory act. During forced expiration, the air being driven from the lung-tissue in front of the heart, the beat becomes more forcible, and its position elevated on account of the descent of the ribs which occurs in expiration.

In view of the influence exerted by respiration and position on the apex-beat the patient, as a rule, should be examined in the erect or sitting posture, while breathing quietly.

Displacement of the Apex-beat.

Displacement to the left may result from :—

1. Hypertrophy and dilatation of the heart (down and to the left.)
2. Pericardial effusion (up and to the left).
3. Chronic diseases of the left lung and pleura, associated with retraction—as fibroid phthisis and pleural adhesions.
4. Abdominal tumors and effusions (up and to the left).
5. The pressure of a pleural effusion on the right side (up and to the left).

Displacement to the right may be caused by :—

1. Chronic disease of the right lung or pleura associated with retraction.
2. Pressure of a pleural effusion on the left side.

Displacement downward may result from :—

1. Hypertrophy and dilatation of the heart, chiefly the left ventricle.
2. Pressure of solid growths in the upper mediastinum.
3. Aneurism of the aortic arch.
4. Enlargement of the liver, causing traction through the central tendon of the diaphragm. (Paul.)

Deformity of the chest may cause displacement in any direction.

Changes in Force and Extent of the Apex-beat.

The force and extent may be increased by :—

1. Hypertrophy of the heart.

2. Excited action of the heart, from drugs, reflex irritation, excitement, or diseases, as exophthalmic goitre.
3. Shrinking of the lungs, as in phthisis.

A weak apex-beat may be noted in :—

1. Healthy people.
2. Degeneration or dilatation of the heart.
3. Pericardial effusion.
4. Emphysema.
5. Shock or collapse.

Abnormal Centres of Pulsation.

Epigastric pulsation may result from :—

1. Excited action of the heart from any cause.
2. Enlargement of the right ventricle.
3. A pulsating aorta noted in certain nervous and anæmic patients.
4. Aortic aneurism.
5. Tumors of the left lobe of the liver resting on the aorta.

Pulsation at the base of the heart may result from :—

1. Aneurism of the aortic arch.
2. Cardiac hypertrophy.
3. Shrinking of the lungs, as in phthisis.

Pulsation in the left axillary region may result from :—

1. Enlargement of the heart.
2. A tense purulent effusion in the left pleural sac (pulsating empyema).
3. Aneurism.
4. Chronic diseases of the left lung and pleura, associated with retraction.

Unnatural pulsation in the carotids may result from :—

1. Excitement of the heart from any cause.
2. Exophthalmic goitre.
3. Anæmia.
4. Valvular disease, especially aortic regurgitation.
5. Aneurism or dilatation of the vessels.
6. Unnatural elasticity of the vessels, noted in certain nervous and anæmic patients.

Jugular Pulsation.

The jugular vein often becomes distended in forced expiration and coughing. Distention of the jugular vein is sometimes noted in adherent pericardium.

A true, rhythmical venous pulsation usually results from tricuspid regurgitation.

A pulsation may be transmitted to the jugular vein from the underlying carotid, but this false pulsation will still continue when light pressure is made on the vein at the root of the neck, while the true venous pulse will cease.

Præcordial Prominence.

Unnatural prominence of the præcordia may result from :—

1. Hypertrophy of the heart.
2. Dilatation of the heart.
3. Pericardial effusion.

PALPATION.

This not only determines the position, force, extent, and rhythm of the apex-beat, but also detects the existence of any fremitus or thrill.

A thrill is a vibratory sensation likened to that received when the hand is placed on the back of a purring cat. Thrills at the base of the heart may result from valvular lesions, atheroma of the aorta, aneurism, and from roughened pericardial surfaces, as in pericarditis.

A presystolic thrill at the apex is almost pathognomonic of mitral stenosis.

PERCUSSION.

This determines the shape and extent of the cardiac dulness.

The normal area of superficial or absolute percussion-dulness (the part uncovered by lung) is detected by light percussion, and extends from the fourth left costo-sternal junction to the

apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum and thence up the left border of the sternum.

The normal area of deep percussion-dulness (the heart projected on the chest-wall) is detected by firm percussion, and extends from the third left costo-sternal articulation to the apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum; and thence up the right border of the sternum to the third rib. The lower level of the cardiac dulness fuses with the liver dulness, and can rarely be determined by percussion.

The area of cardiac dulness is increased in: (1) Hypertrophy and dilatation of the heart. (2) Pericardial effusion. It is apparently increased in shrinking of the lungs, as in phthisis.

The area of cardiac dulness is diminished in: (1) Emphysema. (2) Pneumothorax. (3) Pneumoperieardium (rare). (4) Gaseous distention of the stomach.

AUSCULTATION.

This determines the quality, intensity, and rhythm of the heart-sounds, and detects the presence of any adventitious sounds, as murmurs. The two sounds heard over the heart have been represented by the syllables, "lubb, tup." The first sound (systolic) results from contraction of the ventricle, tension of the auriculo-ventricular valves, and the impact of the heart against the chest-wall, and is synchronous with the apex-beat and carotid pulse. This sound is prolonged and dull. After the first sound there is a short pause, and then follows the second sound (diastolic), which results from the closure of the aortic and pulmonary valves. This sound is short and high-pitched. After the second sound a longer pause follows before the first is again heard.

The Intensity of the Heart-sounds.

Both sounds are accentuated in: (1) Excitement of the heart from any cause. (2) Anæmia. (3) Cardiac hypertrophy. (4) Subjects with thin chest-walls. (5) Consolidation of the lung, as in phthisis and pneumonia.

Accentuation of the aortic second sound results from: (1) Hypertrophy of the left ventricle. (2) High arterial tension, as in arterio-sclerosis and Bright's disease. (3) Aortic aneurism.

Accentuation of the pulmonary second sound results from: (1) Pulmonary obstruction, as in emphysema, pneumonia, and the congestion of the lungs following mitral disease. (2) Hypertrophy of the right ventricle.

Weakness of both sounds is noted in: (1) General obesity. (2) General debility. (3) Degeneration or dilatation of the heart. (4) Pericardial or pleural effusion. (5) Emphysema.

Reduplication of the Heart-sounds.

This is probably due to a lack of synchronous action in the valves of the two sides of the heart, and results from many conditions, but notably from increased resistance in the systemic or the pulmonary circulation, as in arterio-sclerosis of chronic nephritis and in emphysema. It is frequently noted in mitral stenosis and pericarditis.

Adventitious Sounds or Murmurs.

A *murmur* is an abnormal sound heard over the heart or bloodvessels, and may result from: (1) Obstruction or regurgitation at the valves following endocarditis. (2) Dilatation of the ventricle or relaxation of its walls, rendering the valves relatively insufficient. (3) Aneurism. (4) A change in the blood constituents, as in anæmia. (5) Roughening of the pericardial surfaces, as in pericarditis. (6) Irregular action of the heart.

Murmurs produced within the heart are termed endocardial; those produced outside, exocardial; those produced in aneurisms, bruits; and those produced by anæmia, hæmic murmurs.

Hæmic Murmurs.

Hæmic murmurs have the following characteristics: They are soft and blowing in character, usually systolic in time, heard best over the pulmonary valves, transmitted into the

carotids, accompanied with a hum in the veins of the neck, associated with the symptoms of anæmia, and do not cause cardiac hypertrophy.

Pericardial Friction-sounds.

Pericardial murmurs, or friction-sounds, are superficial, rough and creaking in quality, to and fro in time, not transmitted beyond the præcordia, and may be modified by pressure of the stethoscope.

The Aneurismal Murmur, or Bruit.

This is usually loud and booming in character, systolic in time, heard best over the aorta or base of the heart, and is often associated with an abnormal area of dulness and pulsation, and with symptoms resulting from pressure on neighboring structures.

THE PULSE.

The average frequency of the pulse in the adult is between 70 and 80 per minute At birth it is between 130 and 150; in the second year about 100, and so it gradually lessens as the child matures.

Increased frequency of the Pulse (*Tachycardia*).

Habitual frequency is sometimes noted in health. The frequency may be temporarily increased by erect posture, excitement, eating, and the use of stimulants.

Abnormal frequency may result from—(1) Pyrexia. The pulse usually bears a definite relation to the temperature, but in certain diseases, as scarlet fever and septicæmia, it is disproportionately rapid. (2) Exophthalmic goitre. (3) Organic heart-disease. (4) Pressure at the base of the brain sufficient to paralyze the pneumogastrics, as in clot, tumor, and advanced meningitis. (5) Shock. (6) Reflex irritation, as in dyspepsia, ovarian, or uterine disease. (7) An independent paroxysmal neurosis ("Essential Paroxysmal Tachycardia"). (8) Certain drugs—belladonna, nitrites, alcohol, etc. (9) Rheumatoid arthritis (Sansom).

Infrequency of the Pulse (*Bradycardia*).

Physiological slowness is noted in repose, fasting, the puerperium, old age, and habitually in certain people (40 to 60 per minute).

Pathological infrequency is observed in many conditions, notably—(1) In organic heart disease, especially fatty degeneration and fibroid induration. (2) In jaundice. (3) From pressure at the base of brain sufficient to irritate the vagus, as in beginning meningitis. (4) At the close of febrile diseases, as typhoid fever, pneumonia, etc. (5) After the use of certain drugs, as digitalis, aconite, opium, etc.

Irregular Rhythm.

(Arhythmia.)

The Intermittent Pulse.—This *per se* is not significant of any pathological condition. It is habitually noted in certain people, after exercise, eating, excitement, or the use of tobacco, tea, or coffee. It is frequently reflex from gastric, hepatic, uterine, or renal disease. It is common in lithæmia and fatty degeneration of the heart.

There may be a false intermission or infrequency in the radial pulse when the heart fails to transmit all its beats to the wrist. This condition is usually indicative of a weak heart.

The Irregular Pulse.—This has the same significance as the intermittent pulse. It is also very common in myocarditis and valvular disease, especially mitral regurgitation.

Fig. 8.

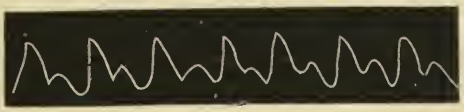
Sphygmogram of the trigeminal pulse.

The Bigeminal and Trigeminal Pulses.—Two or three regular beats followed by a longer pause. They have the same significance as the irregular pulse.

The Pulsus Paradoxus.—One which is more or less suppressed at the close of each full inspiration. It is thought to be due to the compression of the great vessels by inflammatory adhesions, the latter being stretched during the act of inspiration. It is frequently noted in adherent pericardium.

The Dicrotic Pulse.—A pulse in which the main beat is quickly followed by a secondary wave or slight rebound of the vessel. The secondary or dicrotic wave results from a

Fig. 9.



Sphygmogram of a dicrotic pulse.

recoil of the relaxed vessels after the latter have been distended by a sharp ventricular contraction. It is indicative of low arterial tension, and is noted especially in febrile diseases and low states of the nervous system.

Other Variations in the Pulse.

The High-tension Pulse.—One in which the force of the beat is relatively increased. The tension may be roughly estimated by noting the amount of pressure of the fingers that is required to arrest the beat.

A high-tension pulse is observed in many conditions, notably in cardiac hypertrophy, excitement of the heart, chronic nephritis; in cerebral affections irritating the vaso-motor centre, such as apoplexy, tumors, and beginning meningitis; after the use of certain drugs, as digitalis, ergot, and alcoholic stimulants; in chills; in pregnancy; in certain neuroses, as angina pectoris, epileptic and hysterical seizures; and from contraction of the capillaries by irritants generated in the body, as in lithæmia, gout, uræmia.

The Low-tension Pulse.—This is also observed in many conditions, notably in degeneration of the heart, in collapse, in debility, in fevers, and in low states of the nervous system.

Venous Pulse.—A true jugular pulsation is often noted in tricuspid regurgitation. A venous pulse in the dorsum of the hand may be due to (1) forcible propulsion of blood through the capillaries, as in aortic regurgitation with great hypertrophy of the left ventricle; or (2) to extreme relaxation of the arterioles and capillaries, permitting the transmission of the pulse-wave, as in grave cachexia and anæmia.

Asymmetrical Radial Pulses.—May result from: (1) Anomalies in the distribution, size, and division of one of the vessels. (2) Aortic aneurism. (3) An embolus or an atheromatous plate within the vessel. (4) Fractures, luxations, or inflammatory exudations causing compression of the vessel. (5) Compression of one vessel by tumors within or without the thorax.

"Water-hammer Pulse" (*Corrigan's Pulse*).—Characterized by a short, powerful beat, which suddenly collapses. The peculiar pulsation may be distinctly visible, not only in the carotids but throughout the brachial artery. This pulse is diagnostic of aortic regurgitation during the period of compensation, and its force is due to the excessive ventricular hypertrophy and to the large amount of blood expelled with each systole; its sudden recession is due to the incompetent valves failing to support the column of blood.

PALPITATION.

Definition.—A rapid and tumultuous action of the heart perceptible to the patient. Rapidity not perceptible to the patient is not termed palpitation.

Etiology.—It may result from: (1) Reflex irritation, as from gas or acid in the stomach. (2) Excitement, mental or physical. (3) Organic heart disease. (4) Exophthalmic goitre. (5) Over-work, as in the "irritable heart" of untrained recruits. (6) Anæmia. (7) Hysteria. (8) An independent neurosis (Essential Paroxysmal Tachycardia).

DROPSY.

Definition.—An unnatural collection of serous fluid in the tissues or cavities of the body.

Etiology.—Dropsy may result from: (1) Certain chronic visceral affections which bring about venous stasis, as diseases of the heart, liver, and lung. (2) Local obstruction to the venous circulation by emboli, thrombi, tumors, etc. (3) Changes in the composition of the blood, as in anæmia. (4) Changes in the walls of the capillaries, as in Bright's disease. The changes are probably produced by poisons circulating in the blood. (5) Disturbed innervation, as in hysteria and angio-neurotic œdema. In these cases the dropsy is due to either trophic or vaso-motor influences.

GENERAL CYANOSIS.

Definition.—Blueness of the surface from insufficient oxidation of the blood.

Etiology.—Cyanosis results from: (1) Conditions which obstruct the entrance of air, as croup; œdema of the larynx; tumors or foreign bodies in the air-passages; tumors pressing on the air-passages; emphysema; pneumonia; pleurisy; paralysis of the respiratory muscles, as in bulbar palsy; and spasm of the respiratory muscles, as in epilepsy, tetanus, etc. (2) An inability to get blood to the air, as in all forms of chronic heart disease ending in pulmonary congestion.

Congenital Cyanosis is usually associated with stenosis of the pulmonary orifice, an imperfect ventricular septum, or a patulous foramen ovale; it probably results not so much from direct mixture of venous and arterial blood, as from the failure of the blood to reach the lung, or from general venous congestion.

PERICARDITIS.

Definition. — An inflammation of the pericardium, or serous covering of the heart.

Etiology.—(1) Idiopathic, from exposure. (2) Traumatic. (3) Secondary to neighboring inflammations, as pleurisy, phthisis, pneumonia, mediastinal disease. (4) Secondary to some general disease, as rheumatism, Bright's disease, septicæmia, tuberculosis, and the eruptive fevers.

Pathology.—In the early stage the membrane is red, sticky and lustreless; and if the process now ceases, the condition is termed *dry pericarditis.*

If, however, the inflammation continues, an exudate is formed which may be: (1) Sero-fibrinous, (2) fibrinous, or (3) purulent. In the sero-fibrinous form there is little lymph, the exudate being mainly composed of straw-colored serum (a few ounces to several pints), which in favorable cases is gradually absorbed.

In the fibrinous form, serum is scant and the membrane is covered with a butter-like exudate, which subsequently organizes and unites more or less closely the pericardial surfaces, causing adherent pericardium. The adhesions offer resistance to the ventricular contractions and ultimately induce cardiac hypertrophy. In rare instances the fibrinous exudate becomes calcified.

In the purulent form, death usually results; but evacuation of the pus may be followed by union of the pericardial surfaces, and ultimate recovery.

Symptoms.—Moderate fever, præcordial pain and tenderness, dry cough, dyspnœa, and palpitation. The pulse is at first rapid and forcible, but later weak and irregular.

Physical Signs. *First Stage.*—Dry pericarditis.

Inspection.—Negative.

Palpation.—Sometimes a fremitus, from the grating of the roughened pericardial surfaces.

Percussion.—Negative.

Auscultation—A superficial to-and-fro friction-sound, usually heard best at the base of the heart and not transmitted, to any extent, beyond the præcordia.

Second Stage.—Sero-fibrinous effusion.

Inspection.—Bulging of the præcordia.

Palpation.—The apex-beat is feeble or lost. If detected, it is pushed upwards and to the left.

Percussion.—Increased area of dulness, triangular in shape with the base down.

Auscultation.—The heart-sounds are muffled, feeble, and distant.

Purulent effusion yields similar signs, but in addition,—(1) the symptoms of hectic fever, viz: high and irregular fever, sweats, chills, and progressive pallor. (2) Sometimes œdema over the præcordia; and, (3) in doubtful cases, the aspirating needle reveals pus.

Fibrinous pericarditis (Adherent pericardium) is often difficult to recognize, and while the following signs suggest the condition, they are not absolutely diagnostic:—

Præcordial bulging, a weak apex-beat with loud sounds, a systolic retraction or dimpling not only at the apex, but over a large part of the præcordia, a peculiar diastolic collapse of the jugular veins (Friedreich), a feeble apex-beat, with a forcible impulse over the body of the heart (Paul).

With these signs there are often symptoms of heart-failure, such as dyspnœa, dropsy, and cyanosis.

DIAGNOSIS. *Acute Endocarditis.*—The murmur is soft and blowing, not harsh; it is usually single, not to-and-fro; it is somewhat distant, not superficial; it is not necessarily heard best at the base, but at one of the valve points; it is not confined to the præcordia, but is usually transmitted; and it is not followed by the signs of effusion.

Pericardial effusion must be distinguished from *cardiac hypertrophy.* In hypertrophy the area of dulness is increased, but normal in outline; the apex-beat is displaced downwards and to the left, and is forcible; and the sounds are loud and clear.

Pericardial effusion and *cardiac dilatation.*—In dilatation there is no friction-sound; the apex is usually displaced down-

wards, never upwards; the area of dulness is not pyramidal, but extends laterally; the sounds are not muffled, but clear and sharp.

PROGNOSIS.—In the dry and sero-fibrinous forms the prognosis is good under favorable conditions. In the purulent form the outlook is extremely grave. The fibrinous form, though not immediately fatal, is very serious on account of the secondary changes which it induces in the cardiac muscle.

TREATMENT.—Absolute rest. Light diet. Opium is usually required to insure quiet and to relieve pain. When the action of the heart is rapid and irregular, either aconite or digitalis may be administered according to the strength of the pulse.

Local Treatment.—In severe cases apply a few wet cups, leeches, or a blister to the præcordia. In other cases, an ice-bag or poultice may give relief.

Pericardial effusion (Chronic pericarditis).—When the effusion is decided, apply small blisters over the præcordia, administer iodide of potassium (gr. x thrice daily), and encourage diuresis with digitalis or caffeine, and catharsis with saline draughts.

Paracentesis of the pericardium is indicated when the effusion is large and causes dyspnœa, cyanosis, and a weak, rapid pulse, and when the exudate is purulent. The needle should be introduced in the fifth interspace, a little to the right of the point of the normal apex-beat. When the effusion is purulent, a free incision offers a slight, and the only chance of cure.

In adherent pericardium, repeated small blisters may be employed and heart-failure combated with digitalis and similar cardiac tonics.

OTHER AFFECTIONS OF THE PERICARDIUM.

Hydropericardium (Dropsy of the pericardium) results from pericarditis, or from one of the causes of general dropsy, as chronic heart, kidney, or lung disease.

PHYSICAL SIGNS.—The same as sero-fibrinous pericarditis.

Hæmopericardium (Blood in the pericardial sac) results from the rupture of an aneurism, rupture of the heart, traumatism, and cancerous and tuberculous pericarditis.

Physical Signs.—The same as hydropericardium. It is speedily fatal.

Pneumopericardium (Air in the pericardium).—This rare condition results from external wounds, or the rupture of an air-containing organ into the pericardium, as the perforation of a pyo-pneumothorax into the pericardial sac. The entrance of a septic irritant produces pus and the condition becomes a pneumo-pyopericardium.

Physical Signs.—Percussion over the præcordia yields tympany; and auscultation, splashing and metallic sounds.

ENDOCARDITIS.

(Valvulitis.)

Definition.—Inflammation of the lining membrane of the heart. The process is usually confined to the valves.

Varieties.—(1) Exudative, or vegetative endocarditis (Endocarditis verrucosa). This begins as an acute affection, but usually leads to chronic interstitial valvulitis. (2) Sclerotic, or interstitial valvulitis (Chronic endocarditis). (3) Ulcerative, or malignant endocarditis.

Etiology.—*Acute endocarditis* usually results from acute articular rheumatism, one of the infectious fevers, chorea, or septicæmia. Gonorrhœa, tuberculosis, and Bright's disease are occasional causes. At least 40 per cent. of all cases of acute articular rheumatism are complicated with endocarditis. The young are more liable to be attacked than the old. Sixty-two of 73 fatal cases of chorea, collected by Osler, showed endocarditis. Of the infectious fevers, scarlatina and pneumonia are most prone to heart complications.

Chronic endocarditis may be congenital, follow an acute attack, or result directly from alcoholism, syphilis, rheumatism, gout, or Bright's disease. Severe muscular strain sometimes induces it.

Pathology.—Post-natal endocarditis most commonly involves the valves of the left side of the heart.

Pre-natal endocarditis most commonly involves the valves of the right side of the heart.

In the exudative form the valve is red, swollen, lustreless, and studded with numerous bead-like vegetations which are especially marked along its free margins.

These vegetations are composed of proliferated connective-tissue cells, the superficial layers of which have undergone coagulation-necrosis, and are covered with more or less fibrin derived from the blood.

They may be whipped off by the blood-current, and be carried as emboli to distant organs, as the brain, kidney, and spleen; but more commonly, if life is preserved, they are partially absorbed, and the remaining proliferated connective-tissue cells form fibrous tissue, and thus sclerotic valvulitis is secondarily induced.

Sclerotic valvulitis may arise as a primary disease, and is characterized by thickening, curling and puckering of the valve from an overgrowth of fibrous tissue, which is often associated with more or less fatty degeneration of the cells and a deposition of lime salts in their midst.

Symptoms of Acute Endocarditis.—Subjective phenomena are often absent, and auscultation may furnish the only indication of endocarditis, namely, a prolongation of the heart-sound, which later develops into a distinct murmur.

In many cases fever, an irregular and rapid pulse, palpitation, præcordial distress, and dyspnœa are associated symptoms.

Diagnosis.—Chiefly by physical signs. In *pericarditis* the friction-sound is to and fro, superficial, perhaps modified by pressure of the stethoscope, not transmitted much beyond the præcordia, and is followed by signs of effusion.

Prognosis.—In simple endocarditis the prognosis should be guarded. The lesion rarely disappears, and permanent damage to the valve results. Under favorable conditions, however, compensatory hypertrophy of the heart results, and good health may be preserved for an indefinite period.

Treatment.—Absolute rest is of the greatest importance. The original disease which induced the endocarditis should receive appropriate treatment. The application of blisters

is of questionable utility. Cardiac excitement will call for digitalis or aconite, according to the strength of the pulse. Heart-failure must be combated with diffusible stimulants, such as alcohol, nitro-glycerine, and strychnine.

CHRONIC VALVULAR AFFECTIONS.

Period of Compensation.—By compensation is meant an increase in the size and strength of certain cardiac chambers sufficient to enable the arterial system to receive its normal amount of blood, notwithstanding obstruction or regurgitation at one or more of the valves.

The duration of this period is indefinite, and depends largely on the amount of damage sustained by the heart and the hygienic conditions to which the patient is subjected.

During perfect compensation endocarditis is indicated by physical signs, symptoms being entirely absent.

Aortic Stenosis, or Aortic Obstruction.

Definition.—Obstruction to the flow of blood into the aorta from thickening or adhesion of the aortic segments.

Physical Signs. *Inspection.*—If the heart is strong, the apex-beat is forcible, and is noted downward and to the left.

Palpation confirms inspection, and sometimes detects a systolic thrill at the base of the heart.

Percussion may yield an increased area of cardiac dulness, especially to the left.

Auscultation.—A systolic murmur with maximum intensity in the right second intercostal space, and transmitted into both carotid arteries.

Pulse.—During perfect compensation, the pulse is quite normal, but when the heart weakens, it becomes small and slow.

Compensation.—From obstruction to the outflow of blood, the left ventricle becomes hypertrophied.

Sequence.—Mitral regurgitation. Weakening and dilatation of the left ventricle prevent perfect closure of the mitral orifice, and relative insufficiency results.

Aortic Insufficiency, or Aortic Regurgitation.

DEFINITION.—Failure of the aortic valves to prevent a return of blood to the ventricle, from rupture or inflammatory contraction of the segments, or from dilatation of the orifice.

PHYSICAL SIGNS. *Inspection.*—Apex-beat forcible, and displaced downward and to the left. The præcordia may bulge.

Palpation.—Confirms inspection.

Percussion.—Increased area of cardiac dulness, especially to the left.

Auscultation.—A diastolic murmur with maximum intensity in the right second intercostal space, and transmitted down the sternum and towards the apex.

PULSE.—The arteries, especially the carotids, brachials, and radials, pulsate visibly. Palpation detects the "water-hammer," or Corrigan's pulse, *i. e.*, a short, full, and receding pulse.

The extreme cardiac enlargement makes the pulse full, and the prompt leakage back into the ventricle makes it short and receding. Elevation of the arm, during palpation of the radial, makes this pulse more apparent, as the position favors regurgitation. A capillary pulse is sometimes present. It may be noted at the root of the finger-nail by an alternate blushing and paling, synchronous with the heart-beats.

COMPENSATION.—Dilatation and hypertrophy of the left ventricle. Dilatation results from the reception of such a large quantity of blood during diastole, and hypertrophy follows from the increased effort which the ventricle must put forth in emptying itself of this extra quantity of blood.

This extremely dilated and hypertrophied heart has been called the *cor bovinum*, or ox-heart.

SEQUENCE.—Mitral regurgitation. The dilatation and weakening of the ventricle prevent perfect closure of the mitral orifice, and relative insufficiency results.

Mitral Stenosis, or Mitral Obstruction.

DEFINITION.—Obstruction to the flow of blood through the mitral orifice, from thickening or adhesion of the mitral segments.

PHYSICAL SIGNS. *Inspection.*—Apex-beat is not much displaced. There is sometimes bulging over the lower part of the sternum.

Palpation.—A rough presystolic thrill near the apex.

Percussion.—Increased area of dulness, especially to the right.

Auscultation.—A prolonged, rough, churning murmur, presystolic in time, heard most distinctly a little above and to the left of the apex, and not transmitted.

The second sound at the pulmonary cartilage is accentuated from the enlargement of the right ventricle.

PULSE.—During the period of compensation the pulse is small and regular.

COMPENSATION.—From obstruction to the outflow of blood the left auricle becomes enlarged; when it loses power, the blood accumulates in the lung, and to overcome this pulmonary resistance the right ventricle becomes hypertrophied.

There is no strain on the left ventricle, and hence that chamber is not enlarged.

SEQUENCE.—Tricuspid regurgitation. Dilatation of the right ventricle prevents perfect closure of the tricuspid orifice, and relative insufficiency results.

Mitral Insufficiency, or Mitral Regurgitation.

DEFINITION—Imperfect closure of the mitral orifice from rupture or inflammatory contraction of the mitral segments; or from dilatation or weakening of the left ventricle, preventing perfect coaptation of normal valves.

PHYSICAL SIGNS. *Inspection.* — Apex-beat forcible, and noted downward and to the left. The præcordia may bulge.

Palpation confirms inspection.

Percussion.—Increased area of dulness to the right and left.

Auscultation.—A systolic murmur, with maximum intensity at the apex, and transmitted to the left axilla and to the angle of the scapula.

PULSE.—During period of compensation normal, but very irregular when the heart weakens.

Compensation.—The left auricle enlarges from the extra amount of blood that it receives; when it weakens, the lungs become congested and right ventricular hypertrophy follows.

The left ventricle also becomes hypertrophied from its effort to move the large quantity of blood which it receives from the distended auricle during each diastole.

Sequence.—Tricuspid regurgitation. Weakening and dilatation of the right ventricle prevent perfect closure of the tricuspid orifice.

Tricuspid Stenosis, or Tricuspid Obstruction.

This lesion is comparatively rare. It gives rise to enlargement of the heart and a presystolic murmur, which is heard most distinctly at the xiphoid cartilage.

Tricuspid Insufficiency, or Tricuspid Regurgitation.

Definition.—Imperfect closure of the tricuspid orifice from inflammatory shortening of the valves; or, more commonly, from dilatation of the right ventricle secondary to mitral disease or to chronic lung disease.

Physical Signs.—Enlargement of the heart; a systolic murmur, heard most distinctly just above the xiphoid cartilage, and associated with pulsation of the jugular vein, and in bad cases, with pulsation of the liver.

Pulmonary Stenosis, or Pulmonary Obstruction.

This very rare lesion is always congenital, and may be suspected when a systolic murmur is heard most distinctly at the left second intercostal space, and is not transmitted into the vessels of the neck.

Pulmonary Insufficiency, or Pulmonary Regurgitation.

This is very rare, and is always congenital. It produces a diastolic murmur, which is heard most distinctly in the left second intercostal space.

Period of Lost Compensation.—Lost compensation usually results from: (1) Increasing damage to the valves; (2) senility, leading to arterial and cardiac degeneration; (3) some intercurrent disease, throwing additional strain on the heart; and (4) undue physical exertion.

During this period subjective symptoms appear. In cardiac insufficiency, no matter what the original valvular lesion may have been, the organ becomes unable to fill the arteries, and the blood is dammed back in the lungs, and venous congestion of the organs follows.

Symptoms.—Pulmonary congestion produces dyspnœa, asthma, hæmoptysis, and often chronic bronchial catarrh with cough and expectoration.

Hepatic, stomachic, and intestinal congestion produce dyspepsia. Renal congestion produces scanty, albuminous urine, and later nephritis.

General venous congestion produces cyanosis, and dropsy which begins in the feet and mounts upward.

Disturbances of the cerebral circulation produce headache, vertigo, and syncopal attacks.

In aortic disease, especially aortic stenosis, cerebral symptoms are often marked. In mitral disease, pulmonary symptoms are usually marked.

Prognosis of Chronic Valvular Affections.—The extent of damage can never be accurately determined by the quality or intensity of the murmur.

All things being equal, the following is probably the order of gravity in the various valvular lesions: (1) Tricuspid regurgitation, (2) aortic regurgitation (often ending in sudden death), (3) aortic stenosis, (4) mitral stenosis, and (5) mitral regurgitation.

The following are unfavorable conditions: Early life, advanced years, great cardiac enlargement, irregular heart-action, liability to recurring attacks of rheumatism, bad hygienic surroundings, and symptoms of congestion of the lungs, kidney or digestive tract.

In proportion to the absence of these conditions, the prognosis becomes favorable. In many cases life is not materially shortened.

Treatment.—When compensation is perfect, the treatment is purely hygienic.

When there is sudden heart-failure in valvular disease, indicated by orthopnœa and cyanosis, rest should be absolute, hot applications should be applied to the præcordia, and diffusible stimulants administered hypodermically: spirits of ammonia (20–30 minims), whiskey (30–60 minims), sulphate of strychnine (gr. $\frac{1}{30}$, repeated once or twice), and especially nitro-glycerine (1–2 drops of 1 per cent. alcoholic solution) may be so employed; the last, in addition to being a highly diffusible stimulant, has the power of dilating the peripheral bloodvessels. Venesection (10–20 ounces) is often of considerable value in these cases.

When compensation is gradually lost, rest, a light, nutritious diet, and tinct. digitalis (10–20 drops three or four times daily) are the most important therapeutic measures. Tinct. strophan- sometimes succeeds when digitalis fails. Mild laxatives, such as massa hydrargyri (gr. iij–v), greatly influence the absorption of digitalis. When there is moderate dropsy the following pill is very efficient:—

℞ Mass. hydrargyri,
Pulv. digitalis,
Pulv. scillæ, āā gr. xxiv.—M.
Ft. in pil. No. xxiv.
Sig.—One pill thrice daily.

Strychnine is often a valuable adjunct to digitalis, especially when there are indications of fatty degeneration of the heart. When there is anæmia, iron is indicated, and it may be given with digitalis and strychnine, as in the following pill:—

℞ Strychnin. sulph., gr. j;
Pulv. digitalis,
Ferri carb. sacchar., āā gr. xxx.—M.
Ft. in pil. No. xxx.
Sig.—One pill thrice daily.

When there is much bronchitis and dyspnœa, digitalis with ammonia and senega is an efficient combination. (Barlow.) When dyspnœa is marked and the pulse is strong, nitro-glycerine (1–2 drops thrice daily, or gr. $\frac{1}{100}$ thrice daily), if well borne, may be of much service. In extreme dropsy

free catharsis should be induced by compound jalap powder (gr. xx–xxx), or a concentrated solution of Epsom salts (℥ss), and diuresis established by the infusion of digitalis (f℥ ss–f℥j, thrice daily). In persistent anasarca, aspiration of serous sacs and puncture of the legs may be required.

When there is excessive hypertrophy, indicated by præcordial distress and a full, regular pulse, without dropsy, aconite in small doses will prove efficient.

ACUTE ULCERATIVE ENDOCARDITIS.

(Mycotic Endocarditis, Malignant Endocarditis.)

Definition.—A rapidly-destructive form of endocarditis, characterized by necrosis or ulceration of the valves and the deposition of colonies of micrococci.

Etiology.—It may begin as a primary disease, or be engrafted on a simple endocarditis. It may result in the debilitated from overwork or exposure; it sometimes complicates the puerperium; it generally follows septicæmia or one of the specific fevers—such as pneumonia, erysipelas, and scarlet fever. It may be induced by gonorrhœa.

Pathology.—The valves are the seat of ulcers, deep abscesses, and soft, yellowish vegetations, which have undergone partial necrosis. Microscopic examination reveals myriads of micrococci.

Symptoms. 1. *General.*—High and irregular fever, repeated chills, profuse sweats, great prostration, often delirium and stupor, hurried breathing, rapid irregular pulse, brown fissured tongue. Jaundice and diarrhœa are frequently present.

2. *Cardiac Symptoms.*—Præcordial pain, palpitation, and often a blowing murmur at one or more of the valves. Murmurs may be absent.

3. *Embolic Symptoms.*—Peripheral emboli yield a petechial rash; renal embolism may yield bloody urine; splenic embolism may yield a painful spleen; cerebral embolism may yield paralysis.

Diagnosis. *Meningitis.*—Cardiac symptoms, high fever, profuse sweats, and chills will usually separate it from meningitis.

Typhoid Fever.—Abrupt onset, cardiac symptoms, embolic symptoms, sweats, chills, and the absence of the characteristic rash, of the Widal reaction, and of leucocytosis will separate it from typhoid fever.

Malarial Fever.—In endocarditis the *plasmodium malariæ* is not found in the blood.

PROGNOSIS.—Almost invariably fatal. Duration is from a few days to several weeks.

TREATMENT.—Ice-bags to the heart. Light nutritious diet. Stimulants.

ACUTE MYOCARDITIS.

DEFINITION.—Acute inflammation of the heart muscle.

ETIOLOGY.—It is almost always secondary to endocarditis or to pericarditis. As a primary affection of the heart, it may be due to rheumatism, or to one of the infectious fevers.

PATHOLOGY.—The muscle substance is pale, flabby, and friable. Microscopic examination reveals fatty degeneration of the muscle fibres and an infiltration of the connective tissue with leucocytes.

SYMPTOMS.—The symptoms are often masked by the primary disease. Dyspnœa, præcordial pain and distress, a weak, very rapid, small, and irregular pulse, a feeble impulse, and weak sounds suggest the condition.

TREATMENT.—Absolute rest, and the use of cardiac stimulants, like strychnine, caffeine, digitalis, and alcohol.

FIBROID HEART.

(Myo-degeneration of the Heart, Chronic Myocarditis, Indurated Degeneration.)

ETIOLOGY.—This condition is dependent upon atheroma or sclerosis of the coronary arteries. The indirect causes are rheumatism, gout, syphilis, alcoholism, endocarditis and pericarditis.

PATHOLOGY.—The heart is usually hypertrophied or dilated, and is the seat of grayish-white patches, which represent overgrown connective tissue. The papillary muscles,

columnæ carneæ, and the wall of the left ventricle near the apex are the parts most frequently affected.

Arterial sclerosis causes necrosis, and this in turn is followed by a proliferation of the connective tissue.

The fibroid areas sometimes yield to the endocardial pressure and cause aneurism of the heart.

SYMPTOMS.—It manifests the same symptoms as fatty degeneration, viz: dyspnœa, cough, weak and irregular pulse, palpitation, anginoid pains, dropsy, etc.

TREATMENT.—Same as in fatty heart.

HYPERTROPHY OF THE HEART.

DEFINITION.—Enlargement of the heart due to an overgrowth of its muscle.

ETIOLOGY.—It always results from increased work, and this may be due to: (1) Too much blood to be moved from the heart, as in the regurgitant valvular lesions. (2) Obstruction to the outflow of blood at the valves, as in the stenoses; or in the pulmonary or the systemic circulation, as in emphysema and Bright's disease. (3) Resistance to ventricular contraction by pericardial adhesions. (4) Undue physical exertion long continued. (5) Disturbed innervation from drugs, such as tobacco; or from disease, as exophthalmic goitre.

VARIETIES.—(1) *Simple hypertrophy.* Thickened muscle and cavities of normal size. (2) *Eccentric hypertrophy* (hypertrophy with dilatation). Thickened muscle and cavities dilated. (3) *Concentric hypertrophy.* Thickened muscle and cavities diminished in size. Always congenital.

PATHOLOGY.—The average weight of the normal heart is eight or nine ounces; in hypertrophy it may weigh two or three times as much. One or both ventricles may be enlarged; the left is the one more commonly affected. The muscle is firm and of a deep-red color. Histologically the muscle-elements are increased in size and number.

SYMPTOMS.—Unless the hypertrophy is more than compensatory no symptoms result. Extreme hypertrophy is indicated by præcordial distress, palpitation, a strong pulse, and sometimes by the phenomena of cerebral hyperæmia, viz: flushed

face, ringing in the ears, flashes of light. headache, and disturbed sleep.

PHYSICAL SIGNS. *Inspection.*—Præcordial bulging. Forcible impulse. The apex-beat is displaced downward and to the left.

Palpation.—A heaving impulse.

Percussion.—Increased area of cardiac dulness.

Auscultation.—Sounds are dull and loud.

SEQUELÆ.—Apoplexy, fatty degeneration of the heart and subsequent dilatation, valvular disease, and arterial degeneration.

DIAGNOSIS.—*Hypertrophy and dilatation.* These two conditions are commonly associated, but the preponderance of dilatation will be indicated by a feeble fluttering impulse, weak sounds, a weak, irregular, or intermittent pulse, and by symptoms of heart-failure, such as dyspnœa, dropsy, etc.

TREATMENT.—When the hypertrophy is excessive, recommend graduated exercise and a light diet, and employ such sedatives as tincture of aconite (gtt. j–ij thrice daily) or tincture of veratrum viride (gtt. j–ij). The bromides are often valuable adjuncts.

DILATATION OF THE HEART.

DEFINITION.—Enlargement of the heart due to stretching of its walls.

VARIETIES.—(1) Dilatation with thickening of the walls (eccentric hypertrophy), and (2) Dilatation with thinning of the walls.

ETIOLOGY.—Dilatation results from excessive endocardial pressure, as in sudden extreme exertion and in valvular disease, and (2) Impaired nutrition of the cardiac muscle, as in low fevers, valvular disease, and atheroma of the coronary arteries.

PATHOLOGY.—One or both ventricles may be dilated; the right is the one more commonly affected. The condition is usually associated with hypertrophy and fatty degeneration. The muscle may be normal in appearance, but very frequently it is pale and soft.

SYMPTOMS.—So long as the associated hypertrophy keeps pace with the dilatation, no symptoms result; but when dilatation preponderates the following symptoms of venous

stasis appear: dyspnœa, cough, dyspepsia, scanty urine, dropsy, and a feeble, irregular pulse.

Disturbed innervation often causes præcordial distress and palpitation.

Physical Signs.—Apex-beat is diffuse and weak; it may be visible and yet not palpable (Walshe). When the right heart is involved an impulse is noted below the xiphoid cartilage.

Palpation.—A diffuse, feeble, and fluttering impulse.

Percussion.—The area of dulness is increased, especially laterally.

Auscultation.—The sounds are weak and sharp. The first sound loses its muscular element and resembles the second. Co-existing valvular lesions induce murmurs.

Diagnosis.—*Pericardial effusion.* In this condition a friction-sound is frequently present; the outline of dulness is pyriform with the base below, and is not nearly so broad as in dilatation; and the sounds are distant and muffled; and the apex-beat is displaced upwards.

Treatment.—Rest. Light and nutritious diet. Improve the general condition by careful hygienic regulations, and the use of such tonics as iron, quinine, arsenic, and the like. Cardiac tonics, as digitalis, caffeine, strophanthus, and strychnine, are indicated.

In sudden dilatation, use diffusible stimulants, as brandy, ammonia, or strychnine, hypodermically.

FATTY DEGENERATION OF THE HEART.

Definition.—The term fatty heart is applied to (1) fatty infiltration, in which an abnormal amount of fat is deposited in and upon the heart; and (2) to fatty degeneration, in which the cardiac muscle has been metamorphosed into fat.

Fatty Infiltration.

Etiology.—It is a part of general obesity, and hence results from an hereditary tendency, a rich diet, and sedentary habits.

PATHOLOGY.—The heart may be completely imbedded in fat, the grooves along the larger bloodvessels being favorite seats of deposit. Fat is also found between the muscle fibres, although the latter may be perfectly normal.

SYMPTOMS.—Shortness of breath increased by exertion, a weak but regular pulse, præcordial distress, a tendency to pulmonary congestion, with a resulting obstinate bronchitis, and sluggish digestion.

PROGNOSIS.—Favorable.

TREATMENT.—A regulated diet, in which the use of fats, starches, and sugars is restricted. Graduated exercise. The Turkish bath under supervision. Heart tonics, like digitalis and strychnine, are sometimes indicated.

Fatty Degeneration of the Heart.

ETIOLOGY.—(1) It follows hypertrophy in valvular disease. (2) It is frequently due to atheroma of the coronary artery. (3) It is a common result of malnutrition from old age, wasting disease, or anæmia. (4) It is associated with parenchymatous degeneration in the infectious fevers. (5) It results from mineral poisoning, as by arsenic, antimony, phosphorus.

PATHOLOGY.—The muscle is pale, soft, and flabby, and feels greasy to the hand. Microscopic examination reveals a deposition of granular fat in the muscle-fibres.

SYMPTOMS.—When the condition is marked, it is characterized by all the symptoms of heart-failure, namely, dyspnœa, asthma, cough, a weak, irregular pulse, which may be quite rapid or unusually slow, poor digestion, weak heart-sounds, a feeble apex-beat, dropsy, attacks of syncope, and, near the end, Cheyne-Stokes breathing.

Disturbed innervation often causes palpitation, præcordial distress, and attacks of angina pectoris.

There may be associated evidences of atheroma, namely, rigid arteries, and in the cornea, a fatty arcus senilis.

PROGNOSIS.—Unfavorable. Death may occur suddenly on slight exertion.

TREATMENT.—Rest of mind and body. A carefully-regulated diet, which should be light but nutritious. Iron,

quinine, and arsenic are sometimes indicated. In this condition strychnine (gr. $\frac{1}{60}$—$\frac{1}{30}$ thrice daily) is often of great value. Nitro-glycerine (gr. $\frac{1}{100}$ or one minim of the one per cent. thrice daily) may relieve the distressing symptoms. Restlessness, præcordial distress, and insomnia will call for morphine.

In angina, hot applications should be applied to the præcordia, and nitrite of amyl administered by inhalation.

ANGINA PECTORIS.

(Neuralgia of the Heart, Stenocardia.)

Definition.—A symptomatic affection most commonly associated with occlusion of the coronary arteries and degeneration of the myocardium, and characterized by severe paroxysmal pain in the region of the heart.

Etiology.—It usually develops after middle life, and is more common in men than women. The predisposing causes are those of arteriosclerosis—*i. e.*, alcoholism, gout, syphilis, and Bright's disease. In some instances an hereditary tendency has been noted, and not infrequently the attacks have been preceded by prolonged mental anxiety.

False angina (*pseudo-angina pectoris*) is sometimes associated with hysteria, reflex irritation, or the excessive use of tobacco.

Pathology.—Obstruction of the coronary arteries from atheroma or thrombosis, with resultant degeneration of the cardiac muscle, is the condition usually found after death.

Symptoms.—The attacks are usually precipitated by emotional or physical excitement or indigestion, and are characterized by severe pain, radiating from the heart to the shoulder and down the arm (usually the left), a pale, anxious face, a sense of impending death, dyspnœa, and fixation of the body. The pulse is very variable.

The attacks last from a few seconds to several minutes, and may recur at intervals of a few days to several years.

Diagnosis. *Gastralgia.*—The pain does not radiate to the shoulder and thence down the arm; there is no fear of

approaching death, and no fixation of the body; the attack usually appears when the stomach is empty; there is no evidence of organic heart disease.

Pseudo-angina, or Hysterical Angina.—This affection occurs chiefly in women of a neurotic temperament; is unassociated with organic heart disease; usually occurs at night; rarely induces fixation of the body; is of longer duration than true angina; and is associated with emotional excitement.

Prognosis.—Grave. Sudden death is to be expected.

The duration is often long, and in some instances recovery follows. The prognosis is more favorable when the paroxysms are mild, infrequent, unassociated with organic lesions, and brought on by exertion.

Treatment. *The Attack.*—Inhalation of nitrite of amyl (a few drops on a handkerchief) and hot applications to the præcordia. If prompt relief does not follow, morphine sulphate (gr. $\frac{1}{4}$) with atropine sulphate (gr. $\frac{1}{120}$) may be given hypodermically.

The Interval.—Rest of body and mind. A carefully-regulated diet, which should be light but nutritious.

Iodide of potassium (gr. x thrice daily) over a long course has been highly recommended.

Nitroglycerine (gr. $\frac{1}{100}$ to $\frac{1}{50}$) when well borne is sometimes extremely useful in warding off the attacks. Patients may be provided with glass capsules of nitrite of amyl. General tonics, like strychnine, iron, and arsenic, are often indicated.

ANEURISM OF THE AORTA.

Definition.—A circumscribed dilatation of the aorta.

Etiology.—The male sex, middle life, and laborious work are general predisposing factors. The conditions which lead to arterial degeneration, like syphilis, rheumatism, gout, and alcoholism, are potent predisposing causes.

Sudden exertion is commonly the exciting cause.

Pathology.—Aneurisms are divided according to shape into the fusiform, saccular, and cylindrical forms. When all the arterial tunics have yielded, the dilatation is termed a true

aneurism; when the internal tunic alone has ruptured, and blood has escaped between the layers, it is termed a false or dissecting aneurism.

A true aneurism is composed (1) of an external or adventitious sac which results from inflammation and condensation of the surrounding connective tissue; (2) of one or more of the degenerated coats of the vessel; and (3) of a clot, which is often firm and laminated.

The arch of the aorta is the most common seat. About ten per cent. of aortic aneurisms are abdominal.

Thoracic Aneurism.

Physical Signs. *Inspection.*—This often detects an abnormal prominence and pulsation in the upper sternal region.

Dilatation of the superficial veins may also be noted, and in advanced cases the skin over the prominence may be red and glossy.

Palpation.—This often detects an expansile pulsation and a systolic thrill.

If the cricoid cartilage is grasped between the fingers and thumb, and drawn upwards, a pulsation or tug may be transmitted to the trachea.

Percussion.—This occasionally reveals circumscribed dulness and increased resistance.

Auscultation.—If the clot is not too large, the ear may detect a systolic bruit or murmur. Accentuation of the heart-sounds is often noted.

Pulse.—The pulse in one radial may be delayed, and diminished in volume from the diffusion or spending of the current within the sac, or from the partial occlusion of the arterial orifice.

Symptoms. — Dyspnœa results from pressure upon the trachea, bronchi, or recurrent laryngeal nerve, the last causing spasm or paralysis of the vocal cords. Cough is rarely absent, and when due to spasm of the vocal cords it is of a metallic, barking character.

Pain frequently results from pressure on the bones—vertebræ and sternum, or from irritation of neighboring nerves.

Dilatation or contraction of one pupil may result from pressure on the cervical sympathetic, and unilateral sweating of the face is sometimes induced by the same cause.

Difficult swallowing (dysphagia) results from pressure on the œsophagus; and dilatation of the superficial veins, cyanosis, and local œdema may result from pressure upon the deep-seated veins.

Diagnosis.—*A solid tumor* may yield a transmitted pulsation and simulate aneurism, but in the former the pulsation is up and down, not expansile, the impact is less pronounced, the bruit is usually absent, the heart-sounds are not accentuated, there is no tracheal tug, and the health is generally more impaired.

Pulsating Empyema.—A left-sided purulent effusion may transmit a cardiac pulsation, but the latter is not expansile, the dulness is diffuse, the bruit is absent, and the history will suggest pleurisy.

An expansile aorta may simulate aneurism. This condition usually occurs in women of a neurotic temperament, and lacks the bruit and pressure-symptoms.

Prognosis.—Always grave. The average duration is from one to two years. Death may result (1) from rupture externally, or internally into the pericardium, heart, pleural sac, bronchi, lung, or œsophagus; (2) from exhaustion; (3) from heart-failure, for sometimes the aneurism dilates the aortic orifice and thereby causes aortic insufficiency.

Treatment.—Mechanical treatment by ligation of distal arteries, acupuncture, and electrolysis, has not only been unsatisfactory, but has often shortened life.

The treatment commonly employed is a modification of Tufnell's method, and consists in absolute rest in bed for from eight to twelve weeks, with a dry diet, and the administration of iodide of potassium, which is used empirically in doses of ten to twenty grains, thrice daily. When the pulse is very strong, heart sedatives like aconite and veratrum viride may be administered, or venesection cautiously practised. Pain is often temporarily relieved by the iodide, but when it is severe an ice-bag may be applied locally and morphine given hypodermically.

Aneurism of the Abdominal Aorta.

Seat.—It is most frequently located near the cœliac axis.

Symptoms.—It may be recognized by sharp pain in the back, radiating along the spinal nerves, and increased by eating and drinking, by a delay in the femoral pulse, by gastro-intestinal symptoms, and by physical signs similar to those of thoracic aneurism.

Diagnosis.—*An abdominal cancer* may receive a pulsation from the aorta, and simulate aneurism, but in the former, pulsation is not expansile, and is frequently lost when the patient is placed in the knee-breast posture; and there is greater cachexia, and gastro-intestinal disturbance.

The pulsating aorta of nervous women may simulate aneurism, but there are no pressure-symptoms, or distinct tumor, and it is in the sex in which abdominal aneurisms are very uncommon.

Prognosis.—Very grave. Death usually results from rupture.

Treatment.—Same as in thoracic aneurism. Compression of the aorta, the patient having been anæsthetized, has given good results.

ARTERIO-SCLEROSIS.

(Atheroma, Gull and Sutton's Disease.)

Definition.—A thickening of the arteries due to an overgrowth of connective tissue, associated with more or less fatty degeneration and calcification.

Etiology.—Old age, gout, rheumatism, alcoholism, syphilis, lead-poisoning, nephritis, and laborious work are predisposing causes.

Pathology.—The arteries are thickened, tortuous, and rigid. The intima reveals roughened and opaque areas, which are often the seat of calcareous deposits. In extreme cases there may be spots of necrotic softening in the subendothelial tissue, forming "atheromatous abscesses." Microscopic examination shows more or less fatty degeneration of the different coats, and an overgrowth of connective tissue in the intima.

SYMPTOMS. *Circulatory Phenomena.*—Rigidity of the peripheral vessels, a sluggish, high-tension pulse, accentuation of the second aortic sound, palpitation, dyspnœa, anginoid pains, and hypertrophy of the left ventricle.

Renal Phenomena.—The urine is increased in quantity, is pale in color, and of low specific gravity. It may contain a trace of albumin and a few hyaline casts.

Cerebral Phenomena.—Headache, vertigo, disturbed sleep, failure of memory, and tinnitus aurium.

SEQUELÆ.—Cerebral congestion, apoplexy, fatty heart, dilatation of the heart, angina pectoris, aneurism, interstitial nephritis, gangrene of the extremities.

TREATMENT.—A careful regulation of the habits, clothing, and diet. Stimulants must be avoided. Iodide of potassium (gr. v thrice daily) has been recommended for its absorbent effect. Nitroglycerine is sometimes valuable in overcoming the high arterial tension.

DISEASES

OF THE

RESPIRATORY SYSTEM.

THE NOSE.

The Red Nose.—A nose which is permanently and uniformly red generally indicates alcoholism or acne rosacea. A nose which is permanently red and swollen at the extremities, and has a broadened bridge, indicates chronic hypertrophic rhinitis.

Flattening of the Bridge.—This may result from traumatism or tertiary syphilis.

Movement of the Alæ Nasi during Respiration.—Playing of the alæ is occasionally noted in health, but it is generally an indication of some obstruction to the entrance of air. It is frequently observed in spasmodic croup, true croup, laryngeal œdema, capillary bronchitis, and pneumonia.

Nasal Discharge.—Temporary "running from the nose" is a symptom of acute coryza, measles, hay-fever, diphtheria, and influenza. An offensive discharge should suggest nasal diphtheria, or the impaction of a foreign body.

Chronic discharge occurs in chronic rhinitis. In infants, chronic nasal discharge with mouth-breathing ("snuffles") is very suggestive of hereditary syphilis.

The Sense of Smell.—This is tested by holding odoriferous substances before one nostril at a time while the other is closed. Pungent vapors should be avoided, as the irritation which they excite, and not their odor, may lead to their recognition.

The sense of smell is impaired or lost (anosmia) from :—

1. Rhinitis or morbid growths.
2. Affections of the anterior part of the brain, involving the olfactory nerves or bulbs—as injury, tumor, meningitis.
3. Lesions of the olfactory centres.
4. Paralysis of the trigeminal nerve (by inducing dryness of the mucous membrane).
5. Old age.

An increase (hyperosmia) *or a perversion* (parosmia) *of the sense of smell* may occur in hysteria, insanity, and as an aura of epilepsy.

Epistaxis.—Hemorrhage from the nose occurs under the following conditions: (1) Traumatism. (2) Inflammation. (3) Obstructed circulation—as in chronic heart, lung, and liver disease. (4) Blood-dyscrasia—as in scurvy, infectious fevers, hæmophilia, and purpura. (5) Onset of fevers, especially typhoid. (6) Vicarious menstruation. (7) In rarefied atmosphere, as in mountain-climbing. (8) Often without obvious cause.

THE LARYNX.

Spasm of the laryngeal adductors is characterized by intense dyspnœa and occurs in spasmodic croup; in true croup; in ulceration of the larynx; in laryngismus stridulus; in whooping-cough; in tetany; in hysteria; in hydrophobia; in the laryngeal crisis of locomotor ataxia; when foreign bodies have lodged in the larynx; and when aneurisms or mediastinal tumors press on the recurrent laryngeal nerve and irritate it.

Aphonia or loss of voice may occur :—

1. In severe inflammation of the larynx.
2. From hysteria.
3. In centric paralysis of the recurrent laryngeal nerves, as in bulbar palsy and in tumors of the medulla.
4. In peripheral paralysis of the recurrent laryngeal nerve caused by the pressure of an aneurism, mediastinal tumor, or pericardial effusion.
5. From prolonged use of the voice.
6. From the lodgment of foreign bodies.
7. From cicatricial stenosis of the larynx.

Paralysis of the Laryngeal Muscles.

	Causes.	Symptoms.	Laryngoscopic Appearance.
Paralysis of all of the muscles.	Hysteria; bulbar palsy; pressure upon both vagi or spinal accessories.	Aphonia, but no cough or dyspnœa.	The cords are midway between adduction and abduction, and are motionless ("cadaveric position").
Complete unilateral paralysis.	Pressure upon one recurrent laryngeal by an aneurism or tumor.	Voice weak and rough; no cough or dyspnœa.	One cord is moderately abducted and motionless; the other is drawn beyond the median line in phonation.
Complete paralysis of the abductors.	Catarrhal laryngitis; bulbar palsy; pressure on both vagi or recurrents; hysteria.	Voice quite natural; inspiratory stridor and dyspnœa; no cough.	The cords are near together, and brought still closer by inspiration.
Unilateral paralysis of the abductors.	Pressure on one recurrent by an aneurism or mediastinal tumor.	Hoarseness; fatigue after moderate use of the voice; slight dyspnœa.	One cord is near the median line, and is motionless on inspiration.
Complete paralysis of the adductors.	Hysteria; laryngitis; prolonged use of the voice.	Aphonia, but no cough or dyspnœa.	Cords are open and move naturally on respiration, but are motionless during attempted phonation.

RESPIRATION.

Dyspnœa.—Dyspnœa implies difficult breathing with or without an increase in the number of respirations. Dyspnœa which is so severe as to necessitate a sitting posture is termed orthopnœa. Dyspnœa may occur on inspiration, expiration, or both.

Dyspnœa on expiration is chiefly noted in pulmonary emphysema and asthma.

Dyspnœa on inspiration, or on both inspiration or expiration. In this form the base of the chest is retracted during the violent inspiratory efforts.

Its chief causes are: (1) Obstruction in the larynx from spasm, paralysis, false membrane, œdema, or a foreign body. (2) Pressure of an aneurism, tumor, or large glands upon the trachea, bronchi, or recurrent laryngeal nerve. (3) Asthma. (4) Diseases of the lungs, as pneumonia, emphysema, œdema, phthisis, abscess, and gangrene. (5) Pleural effusions. (6) Cardiac disease. (7) Paralysis of the muscles of respiration. (8) Abdominal distention. (9) Anæmia.

The number of respirations per minute. In the healthy male adult the number of respirations is about 18 to 20 per minute. In women and children, breathing is somewhat more rapid. The ratio between respirations and pulse-beats is 1 to 4 or 4.5.

Rapid respirations are noted in excitement; in pyrexia; in inflammatory diseases of the lungs; in anæmia; in certain affections involving the base of the brain; in poisoning from certain drugs which affect the respiratory centre; in hysteria; in painful affections of the respiratory muscles, as pleurodynia, pleurisy.

Infrequent respirations are observed in certain diseases of the brain, as meningitis, tumor, apoplexy; in advanced fatty degeneration of the heart; in certain forms of coma, particularly uræmic and diabetic; in poisoning with certain drugs, especially opium; in obstruction to the air-passages, as in asthma and in laryngeal spasm.

Cheyne-Stokes, or tidal-wave breathing. In this type the respirations gradually increase in rapidity and volume until they reach a climax, then gradually subside and finally cease entirely for from five to fifty seconds, when they begin again. It depends on some disturbance of the respiratory centre the exact nature of which is still undetermined. It is usually a forerunner of death, but cases have been reported in which it has lasted several months.

Its chief causes are: (1) Certain cerebral diseases, as apoplexy, meningitis, and tumor. (2) Advanced cardiac disease, especially fatty degeneration. (3) Certain forms of coma, especially that produced by uræmia, opium-poisoning, and sunstroke.

COUGH.

Cough results from: (1) All diseases of the lungs and bronchi. (2) Many diseases of the larynx. (3) Foreign bodies in the air-passages. (4) Certain infectious diseases, most of which, however, are associated with catarrh, as whooping-cough, measles, influenza. (5) Inhalation of irritating vapors or gases. (6) Reflex causes, such as pressure on the recurrent laryngeal nerve by an aneurism, and uterine and gastro-intestinal affections. (7) Hysteria.

Laryngeal Cough.—This cough has a hard, metallic, ringing intonation, and has been termed "croupy". It is observed in laryngitis; in whooping-cough; in tuberculosis and syphilis of the larynx; when a foreign body has lodged in the larynx; when an aneurism or mediastinal tumor presses on the recurrent laryngeal nerve, and irritates it; and in hysteria.

Dry Cough.—Cough without expectoration is especially observed in the beginning of inflammatory diseases of the bronchi and lungs; in pleurisy; in most chest diseases of early childhood; and from reflex irritation.

Moist, or loose cough occurs in bronchitis, bronchiectasis, convalescent pneumonia, and phthisis.

EXPECTORATION.

Mucoid sputum is noted especially in the beginning of acute bronchitis; in asthma; in the early stage of pneumonia; and in pulmonary œdema. In the last it is very frothy and watery.

Muco-purulent Sputum.—This is observed in subacute and chronic catarrhal affections of the lungs and bronchi, especially in chronic bronchitis, convalescent pneumonia, and phthisis.

Purulent Sputum.—Sputum is rarely composed of pure pus. Expectoration almost entirely purulent is observed in bronchiectasis, in phthisis with cavities, in abscess of the lung, and when an empyema ruptures into the lung.

Prune-juice Sputum.—Expectoration tinged with altered blood so as to resemble prune-juice. It results from retention of the blood in the lung, and is observed in advanced croupous pneumonia, especially low forms, in gangrene of the lung, and in cancer in the lung.

Rusty Sputum.—A rusty and tenacious sputum is strongly indicative of croupous pneumonia.

Sputum containing fibrous shreds is observed in membranous croup, in diphtheria, and in fibrinous bronchitis.

Currant-jelly sputum is indicative of cancer in the lungs.

Fetid sputum usually results from bronchiectasis, advanced phthisis with cavities, gangrene of the lung, and abscess of the lung.

Such sputum when allowed to stand in a conical glass settles in three layers: an upper layer of dirty froth, a middle layer of turbid mucus in which are suspended purulent strings, and a bottom layer of decomposed pus.

Nummular Sputum.—Sputum found in round, flat, coin-shaped masses, which are heavy and sink in water. This sputum is observed in advanced phthisis, in chronic bronchitis, and in bronchiectasis.

THE MICROSCOPY OF SPUTUM.

Elastic fibres are found in the sputum in phthisis, abscess, gangrene of the lungs, and in some cases of bronchiectasis.

Fig. 10.

Elastic Fibres.

The Detection of Elastic Fibres.—Place the sputum which has collected during the night in a glass beaker, and add to it an equal volume of a solution of caustic soda (20 grains to the ounce), and boil over a spirit-lamp, stirring it occasionally with a glass rod. As soon as it boils pour into a conical glass, and add four or five times the amount of cold distilled water. Allow the mixture to stand for two to three hours, and examine the sediment as for tube-casts. (Fenwick.)

Spirals of Mucin.—Tightly-coiled spirals of mucin, which probably represent moulds of the fine bronchioles, were first pointed out by Curschmann in the sputum of asthma. They have also been observed in the sputum of croupous pneumonia.

Charcot-Leyden's Crystals.—These are small transparent octahedral crystals, similar to those found in the blood of leucæmia. They are observed especially in the sputum of asthma. They have also been noted in phthisis, in fibrinous bronchitis, and in acute bronchitis.

Fig. 11.

Charcot-Leyden's Asthma Crystals. (After Riegel.)

Crystals of Fatty Acids.—These occur as fine needles, singly or in bundles, and are often sharply curved near their extremities. They are observed in the sputum of chronic bronchitis, of abscess, and of gangrene of the lungs.

Crystals of Hæmatoidin.—These occur as small yellow needles, rhombic plates or tufts, and are found in sputa which contain altered blood. They may be observed in abscess, gangrene, and cancer of the lungs.

Tubercle Bacilli.—The presence of tubercle bacilli in the sputum is an absolute proof of tuberculosis, but a failure to detect them after one or two examinations is no proof against

phthisis. The bacillus is a fine rod, in length about half the diameter of a red-blood corpuscle, and often slightly bent and beaded. Its detection depends on its power, when stained, of resisting the bleaching effect of acids. To view it successfully, a $\frac{1}{12}$ oil immersion lens is required.

Fig. 12.

Needles of Fatty Acids. (After Strümpell.)

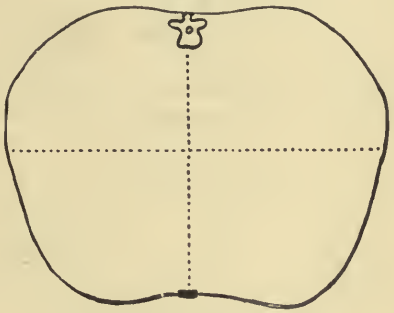
Gabbett's Method.—Select with a clean needle one of the minute caseous masses contained in tubercuIous sputum, spread it out in a very thin film on a cover-glass, dry in the air, and coagulate the albumin in the bacteria by passing the cover-glass, smeared side up, three times through the flame. Cover the specimen with Ziehl's carbol-fuchsin solution (fuchsin 1, alcohol 10, 5 per cent. aqueous solution of carbolic-acid crystals 90), and hold the cover-glass over the flame for a few minutes at such a distance that steam is formed. Wash off the excess of stain in water, and counterstain by treating the preparation for 30 seconds with Gabbett's solution (methyl-blue 2, sulphuric acid 25, water 75). Again wash in water, dry, and mount in Canada balsam. The tubercle bacilli will appear as red rods in a blue field.

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS.

Inspection.

Inspection determines the shape of the chest, any unnatural prominence or depression, the amount of expansion, and any inequality of expansion.

Fig. 13.



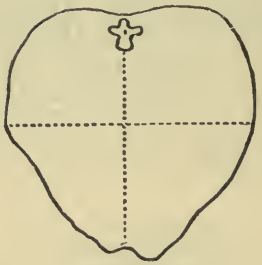
An Outline of the Normal Chest.

Phthisinoid Chest.—The antero-posterior diameter is short; the thorax is long and flat; the ribs are oblique; the scapulæ are prominent; the spaces above and below the clavicles are depressed; and the angle formed by the divergence of the costal margins from the sternum is very acute.

Rachitic Chest.—This may resemble the former, but usually the sides are considerably flattened, and the sternum prominent, so that the term pigeon-breast has been applied to this particular form. The sternal ends of the ribs are enlarged or "beaded," and this characteristic has given rise to the term "rachitic rosary." There is often a circular constriction of the thorax at the level of the xiphoid cartilage.

Emphysematous Chest.—In advanced emphysema the thorax is short and round; the antero-posterior diameter is often as long as the transverse diameter; the ribs are horizontal; the angle formed by the divergence of the costal margin

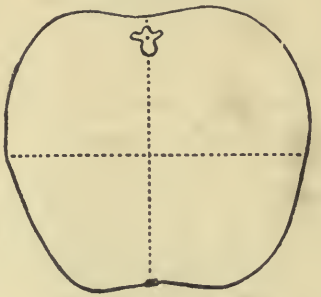
Fig. 14.



Rachitic Chest.

from the sternum is very obtuse or quite obliterated. The term "barrel-shaped chest" is applied to this configuration.

Fig. 15.



Emphysematous Chest.

Local Prominences and Depressions.—An unnatural prominence or depression is often observed over the lower part of the sternum, and is generally congenital. The term funnel-breast or shoemaker's-breast (because it may result from the pressure of tools) has been applied to the sternal depression.

A Unilateral or Local Depression may be due to: (1) Phthisical consolidation. (2) Cavity. (3) Pleurisy with fibrous adhesions.

A Unilateral or Local Prominence may be due to: (1) Pleurisy with effusion. (2) Pneumothorax, hydrothorax,

hæmothorax. (3) An aneurism or tumor. (4) Compensatory emphysema, resulting from impairment of the opposite lung. (5) Cardiac enlargements (left side). (6) Enlargements of the abdominal organs, especially the liver and spleen.

Expansion.—In women and in children, breathing is largely thoracic, or costal; in men and in the old of both sexes, it is largely abdominal, or diaphragmatic.

Restricted abdominal breathing is observed in pregnancy, in abdominal tumors and effusions; in peritonitis; in diaphragmatic pleurisy; in paralysis of the phrenic nerve from pressure or from bulbar disease; and occasionally in the "hysterical abdomen."

Palpation.

Palpation serves to detect any thoracic tenderness, œdema, friction-fremitus, or râles, and to determine the vocal fremitus and amount of expansion.

Thoracic tenderness is observed in pleurisy; in phthisis and pneumonia from being associated with pleurisy; in pleurodynia; in intercostal neuralgia (confined to certain spots); and in surgical affections, like caries and fracture of the ribs; and in contusion and inflammation of the parietes.

Œdema of the chest walls is recognized by "pitting" when pressure is made with the finger. It may be observed in empyema; in deep-seated abscesses of the parietes; after the application of a blister; and in general dropsy.

Friction-fremitus and Rales.—The friction-rub of pleurisy and harsh, sonorous râles can sometimes be detected by palpation.

Vocal, or Tactile Fremitus.—The transmission of the vibrations of the voice to the hand.

In determining the vocal fremitus observe the following precautions: Palpate symmetrical parts of the chest; make firm pressure; when comparing use the same pressure on the two sides; apply the hands as nearly parallel to the ribs as possible; and remember that the fremitus is normally stronger over the right apex.

Vocal fremitus is increased in: (1) Phthisical consolidation. (2) Pneumonic consolidation.

Vocal fremitus is decreased in: (1) Pleural effusions—air, pus, serum, lymph, or blood. (2) Emphysema. (3) Pulmonary collapse from an obstructed bronchus. (4) Pulmonary œdema. (5) Morbid growths of the lung.

Percussion.

Percussion determines resonance, pitch, and resistance.

Immediate percussion is performed by striking the chest directly with the fingers. It is not often employed, except over the clavicles, where the bones themselves act as pleximeters.

Mediate percussion is performed by using the fingers of one hand as a plessor, and those of the opposite hand as a pleximeter; or by using a piece of ivory, glass, or hard rubber as a pleximeter, and a small hammer as a plessor.

The use of the fingers alone is preferable, for only in this way can resistance be determined.

In percussion the following precautions should be observed: Place the finger which is being used as a pleximeter firmly against the chest, and preferably parallel to the ribs; make the finger which is used as plessor strike the one on the chest perpendicularly; fix the forearm, and use no more force than can be obtained from a gentle swing of the wrist. When possible, percuss all parts of the chest anteriorly and posteriorly; percuss both in inspiration and in expiration. In comparing the two sides, be sure to percuss symmetrical parts.

Normal Resonance.—On the right side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward to the upper border of the sixth rib in front, and to a line drawn through the tenth spinous process posteriorly.

On the left side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward, within the mammary line to the third rib, outside of the mammary line to the tenth rib, and posteriorly to a line drawn through the tenth spinous process.

Hyper-resonance is observed in the following conditions: (1) Pneumothorax. (2) Cavities—tuberculous or bronchiectatic. (3) Emphysema. (4) Lowered pulmonary tension in

the initial stage of pneumonia and above a pleural effusion (Skoda's resonance). (5) Flatulent distention of the stomach or colon (frequently observed over the left base).

A tympanitic note is a hollow, drum-like sound like that which is normally obtained by percussing the larynx or empty stomach. The above conditions are also capable of producing tympany.

The cracked-pot sound, or *bruit de pôt fêlé,* is a modified tympany, and can be simulated by percussing over the cheek when the mouth is partially open. It may be normally heard over the chest of a crying infant (Walshe). In the adult it usually indicates a cavity which has a free communication with a bronchus. It is best detected by keeping the ear near the open mouth of the patient while percussing.

Dulness or flatness is recognized in the following conditions: (1) Phthisical consolidation. (2) Pneumonic consolidation. (3) Pleural effusions of all kinds, except air. (4) Collapse of the lung. (5) Congestion and œdema of the lung. (6) Enlargement of the liver or spleen (at the bases). (7) Morbid growths in the lung.

Pitch.—Pitch depends largely upon the volume of air, upon the tension of the walls of the cavity, and upon the size of the opening which communicates with the cavity. The less the air, the greater the tension, and the smaller the opening, the higher will be the pitch of the note. It is obvious, therefore, that conditions which are associated with hyper-resonance may yield either a high- or a low-pitched note. In beginning phthisical consolidation, the note over the affected apex is higher pitched; but it must be borne in mind that normally the note over the right apex is higher pitched than that over the left.

Resistance.—The greater the dulness the greater will be the resistance; hence there is always more resistance over a large pleural effusion than over a pneumonic or phthisical consolidation.

Auscultation.

Auscultation determines the character of the breathing and of the vocal resonance, and detects adventitious sounds, like râles.

In *immediate auscultation* the ear is placed directly over the chest, a soft towel only intervening.

In *mediate auscultation* the sounds are transmitted through a stethoscope, which should be applied to the bare chest.

In *auscultation* observe the following precautions: Do not exert much pressure with the stethoscope; when the chest is covered with hair moisten the latter, otherwise it will produce friction-sounds resembling râles. When possible, auscult all over the chest, anteriorly and posteriorly; auscult on quiet breathing, on full inspiration, on full expiration, and after coughing. In comparing the two sides auscult symmetrical parts.

Normal Respiration.—Vesicular breathing is heard over the body of the lungs, and is characterized by a soft, breezy inspiration and a short, low-pitched expiration. Normally, expiration is not more than one-third as long as inspiration. Auscultation over the trachea, or over the main bronchi in the interscapular space, yields bronchial breathing, *i. e.*, harsh breathing with prolonged high-pitched expiration.

Modifications of the respiratory murmur. *Puerile Breathing.*—This type is heard normally over the lungs of children; it is loud, and expiration is higher pitched than in vesicular breathing, and almost as long as inspiration.

Exaggerated Breathing.—This type has almost the same peculiarities as puerile breathing, and is heard over a lung that is doing extra work necessitated by some impairment of its fellow.

Bronchial or Tubular Breathing.—Harsh breathing, with a prolonged high-pitched expiration, which has sometimes a tubular quality. Bronchial breathing is heard over: (1) Phthisical consolidation. (2) Pneumonic consolidation. (3) Lung which is compressed. (4) Rarely over a lung which is infiltrated with a morbid growth.

Amphoric and Cavernous Breathing.—These two are almost identical; the sounds are loud, and expiration is prolonged and hollow. The pitch of amphoric breathing is a little higher than that of cavernous. Amphoric breathing may be imitated by blowing over the mouth of an empty jar.

Amphoric or cavernous breathing may be heard in the fol-

lowing conditions: (1) Phthisical or bronchiectatic cavities. (2) Pneumothorax, when the opening in the lung is patulous. (3) Areas of consolidation near a large bronchus. (4) Sometimes over lung compressed by a moderate effusion.

Asthmatic Breathing.—Harsh breathing with a prolonged wheezing expiration. It may resemble bronchial breathing, but, unlike the latter, it is heard all over the chest.

The Breathing of Emphysema.—Weak breathing, with prolonged low-pitched or inaudible expiration.

Cogged-wheel, or Jerky Breathing.—The respiratory murmur is not continuous, but is broken into waves. It is not indicative of any special disease, but it is frequently observed in bronchitis and in incipient phthisis.

Weak or Shallow Breathing.—This is noted: (1) When the chest-walls are thick. (2) In the old and feeble. (3) In emphysema. (4) In pleural effusion. (5) In incipient phthisis. (6) In painful affections of the chest, like pleurodynia and beginning pleurisy. (7) In pulmonary œdema.

Vocal Resonance.—The vibrations of the voice transmitted to the ear.

Vocal resonance is normally more marked over the right apex. It is *abnormally increased* in: (1) Pneumonic consolidation. (2) Phthisical consolidation. (3) Cavities which freely communicate with a bronchus.

Vocal resonance is diminished or absent in: (1) Pleural effusions—air, pus, serum, lymph, or blood. (2) Emphysema. (3) Pulmonary collapse. (4) Pulmonary œdema.

Bronchophony.—Extreme exaggeration of the vocal resonance; the sounds, but not the words, are transmitted. It is especially noted over marked consolidations and over certain cavities.

Pectoriloquy.—The distinct transmission of articulate speech to the ear; the words appear to emanate from the spot which is ausculted.

Pectoriloquy is heard over: (1) Cavities which communicate with a bronchus. (2) Areas of consolidation in the neighborhood of a large bronchus. (3) Pneumothorax, when the opening in the lung is patulous. (4) Some pleural effusions.

Ægophony.—A modified bronchophony, characterized by a trembling, bleating sound. It is usually heard over slight

pleural effusions near the upper border of dulness, especially near the inferior angle of the scapula.

It is occasionally heard in beginning pneumonia.

Adventitious Sounds. *Râles, or Rhonchi.*—These are abnormal sounds which replace or accompany the respiratory murmur.

- Pulmonary râles
  - Vesicular = Crepitant.
  - Bronchial
    - Dry
      - Sonorous.
      - Sibilant.
    - Moist
      - Subcrepitant.
      - Bubbling.
      - Gurgling.
- Extra-pulmonary râles = Pleuritic friction-sounds.

Crepitant Râles.—These are very fine râles, and are heard at the end of inspiration. They may be simulated by rubbing a lock of hair between the fingers. They have been especially associated with the first stage of croupous pneumonia, and it has been supposed that they were due to the forcible separation of adherent vesicular walls. Râles very similar to, if not identical with these, are heard in capillary bronchitis and in pulmonary œdema.

Dry râles are probably produced by the presence of viscid secretion in the tubes; they have a more or less whistling, musical, or squeaking intonation. They are heard particularly in bronchitis and asthma. Sibilant râles are whistling and high pitched; sonorous râles have a humming quality and are lower pitched. Dry râles may be heard on inspiration, expiration, or both.

Moist râles result from the presence of liquid in the tubes; the thinner the liquid and the larger the tube, the coarser will be the râles. They may be heard on inspiration, expiration, or both.

Subcrepitant, or crackling râles are fine moist râles, and heard in all conditions which are associated with liquid in the smaller tubes, as bronchitis, capillary bronchitis, pulmonary œdema, and beginning phthisis.

Bubbling râles are coarser than subcrepitant; and are heard in bronchitis, in resolving croupous pneumonia, over phthisical deposits which are softening, and over small cavities.

Gurgling râles are very coarse and resemble the bursting of large bubbles. They are heard over large cavities which contain fluid, and in the trachea in the so-called "death-rattle."

Friction-sounds are produced by the rubbing together of roughened pleural surfaces. They may be heard both in inspiration and expiration, and often resemble subcrepitant râles, but they are more superficial and localized than the latter, and are not modified by cough or deep inspiration.

A roughened pleura in the neighborhood of the heart may produce a friction-sound of cardiac rhythm, and one which will still continue when the breath is held; under other conditions pleural friction-sounds cease when respiration is suspended.

Metallic Tinkling.—This name is applied to silvery or bell-like sounds which are heard at intervals over a pneumo-hydrothorax or large cavity. Speaking, coughing, and deep breathing usually induce them. Care must be taken not to confound them with similar sounds produced by the presence of liquid in a distended stomach.

Succussion-splash, or Hippocratic Succussion.—This is a splashing sound produced by the presence of air and liquid in the chest. It may be elicited by gently shaking the patient while auscultating. It nearly always indicates either a hydro- or a pyo-pneumothorax, although it has been detected over very large cavities.

Air and liquid in the stomach produce a similar sound.

Mensuration.

In measuring the sides of the chest observe the following precautions: Measure from the middle of the sternum to the spinous processes; measure both sides after inspiration and after expiration; apply the tape with equal firmness to the two sides. In comparing, measure corresponding levels, and remember that the right side is from half an inch to an inch greater in circumference than the left.

The conditions which render one side more prominent than the other have already been considered.

CORYZA.

(Acute Rhinitis, Cold in the Head.)

DEFINITION.—An acute inflammation of the nasal cavities.

ETIOLOGY.—Exposure to cold drafts and to wet, especially when the body is overheated, is a common cause. It may be excited by the inhalation of irritating vapors or dust. It is an expression of iodism. It is a symptom of certain infectious diseases—especially syphilis, measles, and influenza.

PATHOLOGY.—The mucous membrane is red and swollen. In the first stage there is no secretion, but later irritating, watery mucus flows from the nose and excoriates the lip; this in time is followed by a copious muco-purulent discharge.

SYMPTOMS.—The disease is ushered in with chilliness, *malaise*, fulness in the head, and sneezing. The nasal chambers are obstructed, so that the patient is obliged to breathe through his mouth. At first there is no secretion, but in twenty-four or forty-eight hours a watery discharge is established, which later becomes muco-purulent. Slight fever and its associated symptoms are commonly present. The duration is from a few days to two weeks.

COMPLICATIONS.—The disease is often accompanied with conjunctivitis, pharyngitis, laryngitis, and catarrh of the Eustachian tube and middle ear which results in temporary deafness.

PROGNOSIS.—Favorable.

TREATMENT.—In the early stage a cold in the head can frequently be aborted by the use of hot drinks, a laxative, moderate doses of quinine, and the application of menthol to the nasal chambers. Some crystals of menthol may be placed in a wide-mouth bottle, and their vapor inhaled for from ten to twenty minutes several times during the day. A spray of menthol may be employed :—

℞ Menthol, ʒj;
Ol. amygd. dulcis, *vel* benzoinal, fʒiij.—M.
Sig.—Spray into the nose several times daily.

Cocaine is often efficient in allaying the fulness and distress; a four per cent. solution may be applied to the nose on a pledget of cotton or by means of a camel's-hair brush.

When the symptoms are severe Dover's powder (gr. v) may be given in combination with quinine (gr. v) thrice daily.

CHRONIC NASAL CATARRH.

(Chronic Rhinitis.)

DEFINITION.—A chronic inflammation of the nasal mucous membrane, characterized by increased secretion and impairment of the sense of smell.

ETIOLOGY.—Repeated attacks of acute coryza, impure air, the continual inhalation of irritating dusts or vapors, lowered vitality, and congenital or acquired obstruction of the nasal chambers are causal factors. It is also an expression of syphilis.

VARIETIES.—Two varieties have been recognized: Chronic hypertrophic rhinitis and chronic atrophic rhinitis.

Hypertrophic Rhinitis. SYMPTOMS.—A thick mucous discharge from the nose; great liability to attacks of acute coryza; obstruction of one or both nasal cavities, causing mouth-breathing; a nasal intonation of the voice; frontal headache; and impairment of the sense of smell.

Symptoms of catarrh of the neighboring organs are frequently present. The most common of these are: dryness of the throat and hawking from pharyngitis; deafness from catarrh of the middle ear; and watering of the eyes from catarrhal occlusion of the lachrymal canal.

Inspection.—The bridge of the nose is frequently flattened, and the alæ are thickened and red; the mucous membrane is red and the cavities are more or less occluded from hypertrophy of the cavernous tissue covering the turbinated bones. In advanced cases exostoses from the bony framework are sometimes noted.

PROGNOSIS.—Under judicious and persistent treatment the affection is curable.

TREATMENT.—The naso-pharynx must be kept clean by

means of antiseptic douches or sprays; Dobell's solution (see page 31) or the following may be employed for this purpose:—

℞ Sodii boratis,
Sodii bicarbonatis, āā ʒss;
Sodii benzoatis,
Sodii salicylatis, āā gr. ij;
Sodii chloridi, gr. vij;
Eucalyptol, thymol, āā gr. j;
Menthol, gr. ss;
Olei gaultheriæ, gtt. j;
Glycerini, fʒss;
Alcoholis, fʒj;
Aquæ, q.s. ad Oj.—M.

Mild astringent sprays are often useful, and sulphate of zinc or sulphate of copper (five to ten grains to the ounce) may be employed for this purpose.

Tonics like cod-liver oil, hypophosphites, iron, arsenic, and strychnia are often indicated.

To effect a cure the naso-pharynx must be unobstructed; hypertrophies and exostoses must be removed and deviations of the septum corrected by surgical means.

Atrophic Rhinitis. (*Ozæna.*) Symptoms.—A sense of dryness in the nose and throat; a thick purulent discharge, or the expulsion of discolored crusts; an offensive, putrid odor, which has given rise to the term ozæna; impairment of the sense of smell. The general health is always poor; such patients are usually thin and anæmic.

Inspection.—The chambers are large; the mucous membrane is pale, dry, and glazed; adherent scabs are generally present. In advanced cases, ulceration and necrosis are observed.

Prognosis.—Perfect cure is rarely obtainable; but treatment may effect great improvement.

Treatment.—Crusts must be removed and the nasal chambers kept clean with antiseptic sprays or douches. Stimulating applications are useful, and solutions of nitrate of silver, sulphate of iron, or sulphate of zinc may be employed. A 30 per cent. solution of lactic acid is also recommended. Ebstein uses tampons soaked in balsam of Peru. When there is much purulent discharge a 20 per cent. mixture of ichthyol in cosmoline is very efficient. General tonics like cod-liver oil, hypophosphites, iron, arsenic, etc. are indicated.

ACUTE CATARRHAL LARYNGITIS.

Definition.—An acute catarrhal inflammation of the larynx, characterized by hoarseness, hard cough, and painful deglutition.

Etiology.—Improper use of the voice; exposure to cold and wet; the inhalation of irritating dusts or vapors; the impaction of foreign bodies are its common causes. It is also an associated condition in certain infectious diseases, like whooping-cough, measles, diphtheria, and influenza.

Pathology.—The mucous membrane is red, swollen, and injected.

In grave cases the tissues may be markedly œdematous.

Symptoms.—Hoarseness of the voice or aphonia; hard, ringing cough; pain in the throat increased by speaking, coughing, and swallowing; expectoration, which is first scanty and later muco-purulent; fever and its associated symptoms. In sensitive people, and especially in children, paroxysms of croupy cough and dyspnœa (false croup) may result from spasm of the vocal cords; and when there is much œdema, dyspnœa or asphyxia will be a prominent feature.

Inspection.—The mucous membrane of the laryngeal walls and vocal cords is red and swollen. In grave cases the tissues are highly œdematous.

Prognosis.—In simple laryngitis without œdema the prognosis is altogether favorable. The attack usually lasts from a week to ten days. When there is œdema of the larynx, indicated by dyspnœa or asphyxia, the prognosis is grave.

Treatment.—The patient should be confined to his room and preferably to bed. The temperature of the room should be 70° or 75°, and the atmosphere should be moistened by the generation of steam.

Iodine, or in severe cases an ice-bladder, should be applied to the throat. The inhalation of medicated vapors is decidedly useful, and one of the following may be employed: Lime-water, Dobell's solution, wine of ipecac (diluted with two volumes of water), or the menthol mixture mentioned in the treatment of acute coryza.

Internal Treatment.—A saline laxative may be administered at the beginning, and followed by one of the following sedative mixtures: Dover's powder (gr. v) with quinine (gr. v) thrice daily, or:—

℞ Potassii citratis,
Potassii bromid., āā ʒij;
Apomorph. hydrochlor., gr. ½;
Aquæ et syr. sarsaparillæ comp., āā fʒiss—M.
Sig.—A teaspoonful every two hours to a child of five years.

Or—One of the following tablets devised by Dr. Seiler:—

℞ Potass. chlor.,
Potass. bromid.,
Pulv. ext. glycyrrhizæ, āā ʒj;
Tinct. ferri chlor., fʒss;
Sacchar., etc., q. s.—M.
Ft. in trochisci No xx.
Sig.—One every three or four hours.

Œdema of the larynx, indicated by extreme dyspnœa, will require scarification of the mucous membrane or tracheotomy.

CHRONIC LARYNGITIS.

Simple Chronic Catarrhal Laryngitis. Symptoms.—Tickling in the throat, huskiness of the voice, fatigue and pain after moderate use of the voice, and the expectoration of viscid mucus are the usual symptoms.

Laryngoscopic examination reveals redness of the mucous membrane and sometimes slight ulcerations.

Treatment.—The patient must learn to use the voice properly; sounds must be expelled by the abdominal muscles and diaphragm, and not by the muscles of the throat. Flannel protectors should be avoided, and the application of cool water to the neck, night and morning, instituted in their stead. Tonics are generally indicated. Expectorants which are eliminated by the respiratory mucous membrane are useful; and one of the following may be employed: Terebene (gtt. v on sugar), oleoresin of cubebs (gtt. x–xx on sugar), oil of eucalyptus (gtt. v in capsule).

Topical Treatment.—A faradic current to the neck is often beneficial; medicated solutions should be applied to the larynx by means of a brush or atomizer. The following are the remedies commonly employed: Nitrate of silver, chloride of ammonium, chlorate of potassium, sulphate of zinc, and tincture of benzoin.

Tuberculous Laryngitis.—This is nearly always secondary to pulmonary tuberculosis, but it occasionally occurs as a primary affection.

Symptoms.—Hoarseness of the voice or aphonia; pain in the throat increased by coughing, speaking, or swallowing; and hacking cough are the usual symptoms.

Laryngoscopic Examination.—The mucous membrane is pale and thickened; the arytenoid cartilages are considerably swollen; small, irregular, shallow ulcers with gray bases are frequently noted, particularly in the inter-arytenoid space.

Treatment.—Remedies must be directed to the primary pulmonary disease. Local applications are required to relieve the pain. Powders of iodoform or morphine may be dusted on the ulcers, or a solution of nitrate of silver, of cocaine, or of menthol may be applied by means of a laryngeal brush.

Syphilitic laryngitis may manifest itself in catarrhal inflammation, or mucous patches, but the most common expression is a gummatous infiltration, which breaks down, ulcerates the cartilages, and ultimately leads to cicatrization and deformity.

Symptoms.—Hoarseness of the voice, hacking cough, and some difficulty in deglutition. Subjective symptoms are often absent, though examination may reveal extensive lesions.

Laryngoscopic Examination.—Deep ulcers with raised edges, often symmetrically arranged. Necrosis of the cartilages results in advanced cases.

Diagnosis.—The history, the presence of other syphilitic lesions, the deep symmetrical ulcers, the effect of treatment, and the absence of marked pain and of pulmonary lesions will serve to distinguish it from tuberculous laryngitis.

Treatment.—The system should be rapidly brought under the influence of antisyphilitic remedies; for this purpose mer-

curial inunctions may be employed, and iodides and mercurials given internally :—

℞ Hydrarg. chlor. corros., gr. j ;
Potass. iodidi, ʒij–ʒiv ;
Syr. sarsaparillæ comp., fʒjss ;
Aquæ, q. s. ad fʒiij.—M.
Sig.—A teaspoonful twice daily after meals.

Local applications, carefully applied by the aid of the laryngoscopic mirror, are also required. Iodoform, or acid nitrate of mercury (1 to 5 of water), may be selected for this purpose.

When the laryngeal movements interfere with healing, tracheotomy should be performed. The same operation or mechanical dilatation is sometimes required for the resulting cicatricial stenosis.

SPASMODIC CROUP.

(False Croup.)

Definition.—Spasm of the vocal cords, excited by catarrh of the larynx.

Etiology.—The attacks usually occur in young children, and are induced by the causes of catarrhal laryngitis.

Symptoms.—Generally there has been a little hoarseness and cough during the day, and at night the child is awakened from sleep by a severe paroxysm of suffocative cough. The latter has a peculiar, hard, metallic quality, and is associated with the evidences of dyspnœa, namely : Anxious face, dilating nostrils, prominent sterno-cleido-mastoids, and retraction of the base of the chest with each inspiratory effort. During the paroxysm the skin is hot and the pulse is tense and rapid. In from a few moments to an hour the cough ceases, free perspiration follows, and the child falls to sleep.

Two or three similar attacks may occur in the same night, but on the following day the child appears quite well. A recurrence of the seizures for several successive nights is not infrequent.

Diagnosis. *Laryngismus Stridulus.*—This is a pure neurosis, and is often associated with the rachitic diathesis. The paroxysms resemble those of false croup, but are associated

with a peculiar crowing inspiration, and lack catarrhal symptoms, such as hoarseness and cough.

Prognosis.—Always favorable.

Treatment.—A sponge moistened with hot water may be applied to the throat, or the child may be placed in a hot bath. If these simple measures fail, an emetic will almost invariably bring relief. Wine of ipecac (ʒj) or turpeth mineral (gr. iij–v) may be selected. Subsequent treatment should be directed to the laryngeal catarrh.

MEMBRANOUS CROUP.

(Croupous Laryngitis, True Croup, Pseudo-membranous Laryngitis.)

See *Laryngeal Diphtheria.*

LARYNGISMUS STRIDULUS.

(Spasm of the Glottis, "Child-crowing.")

Definition. — A paroxysmal neurosis, characterized by spasm of the adductors of the larynx, and not excited by any local inflammation.

Etiology.—Early life (within the first two years), male sex, and the rachitic diathesis are the predisposing causes. The discharge of motor force apparently arises in the medulla (bulbar epilepsy), and may be excited by reflex irritation, as in teething and gastro-intestinal disorders. Some regard it as a symptom of tetany.

Symptoms.—The attacks often occur on waking from sleep, and are characterized by a sudden arrest of breathing and tonic muscular spasms. The face is pale, and later cyanosed; the eyes are rolled up; the body is arched; the thumbs are turned into the palms; the legs are extended, and the soles turned inward. In a few seconds the spasm relaxes, and air is drawn through the glottis with a shrill, crowing sound.

The seizures vary greatly in frequency; several may occur in a day, or they may be weeks apart.

Diagnosis.—The intermittent character of the affection;

the peculiar crowing inspiration; the absence of fever, cough, and hoarseness will serve to distinguish laryngismus from *croup*.

PROGNOSIS.—Favorable. In the very young death may result from suffocation.

TREATMENT. *The Paroxysm.*—Cold water may be dashed on the face and head, or a few drops of nitrite of amyl or chloroform may be placed on a handkerchief and held before the nose.

The Interval.—Careful search should be made for some exciting cause; the gums may require lancing, or the gastro-intestinal tract may demand attention. The child should be placed under the best hygienic conditions. The food should be plain and nutritious; tonics, like cod-liver oil, malt, hypophosphites, and arsenic, are generally indicated. The bromide of potassium is an efficient antispasmodic, and may be advantageously combined with antipyrin:—

℞ Antipyrin, gr. xxiv–xlviij;
Potass. bromid., ʒiss–ʒij;
Syr. aurant. cort., fʒij;
Aquæ, q.s. ad fʒiij.—M.

Sig.—A teaspoonful thrice daily.

ŒDEMA OF THE LARYNX.

(Œdema of the Glottis.)

DEFINITION.—An infiltration of serous fluid into the submucous tissue of the larynx.

ETIOLOGY.—It occasionally results from severe attacks of catarrhal laryngitis. It may be induced by severe inflammation of neighboring organs—as the tonsils, parotid glands, and pharynx. It may be a complication of some acute infectious disease—like diphtheria, scarlet fever, or facial erysipelas. It is sometimes associated with ulcerative affections of the larynx, like tuberculosis and syphilis. It may be excited by the irritation of burns, scalds, or caustics. It occasionally occurs abruptly in the course of Bright's disease.

PATHOLOGY.—The connective tissue of the larynx is infiltrated with a serous or sero-purulent fluid. The mucous membrane is tense and changed in color.

Symptoms.—Hoarseness of the voice, and later aphonia; extreme dyspnœa, at first on inspiration but later on expiration also; stridulous respiration; barking cough; and the evidences of dyspnœa, namely: Anxious face, protruding eyes, blue lips, prominent sterno-cleido-mastoids, and retraction of the base of the chest. When the epiglottis is involved the swelling can be detected by the finger in the throat.

Laryngoscopic Examination. — The mucous membrane is swollen and of a reddish-purple color. The epiglottis may resemble a round translucent tumor. In infraglottic œdema the upper part of the larynx may appear normal, but swollen and œdematous membrane is seen projecting through the glottis. The vocal cords are rarely affected.

Prognosis.—Extremely grave.

Treatment.—When the symptoms are not urgent, leeches or blisters may be applied over the larynx, and astringent solutions (tannic acid or alum) sprayed on the œdematous tissues. When the symptoms persist, the parts should be scarified, and if this fails to relieve the dyspnœa, tracheotomy should be performed.

BRONCHITIS.

Definition.—An inflammation of the bronchial tubes, characterized by substernal soreness, cough, muco-purulent expectoration, and dry and moist râles.

Varieties.—(1) Acute catarrhal bronchitis. (2) Chronic catarrhal bronchitis. (3) Fibrinous bronchitis.

Acute Catarrhal Bronchitis

Etiology.—A cold, damp climate; changeable weather; occupations which necessitate confinement, or the inhalation of irritating dusts or vapors; debility; the gouty diathesis; and chronic heart disease are general predisposing factors.

Exposure to cold and wet, particularly when the body is overheated, or the inhalation of irritating gases or dusts is the

usual exciting cause. Acute bronchitis is also an associated condition in certain infectious diseases, especially measles, whooping-cough, typhoid fever, and influenza.

Pathology.—In most cases the trachea and large tubes only are affected. The mucous membrane is red, swollen, injected, and more or less covered with tenacious muco-pus.

Microscopic examination reveals desquamation of epithelium and infiltration of the submucous tissues with leucocytes.

Symptoms.—Chilliness; *malaise;* a sense of soreness and constriction behind the sternum, which is increased by coughing; slight fever (100°–102°) with its associated symptoms; cough at first dry and painful, but later accompanied by muco-purulent expectoration which becomes quite free as the inflammation subsides.

Physical Signs.—Inspection, palpation, and percussion usually give negative results.

Auscultation at first reveals sibilant and sonorous râles on both sides of the chest, and in the second stage, when secretion is established, moist râles.

Diagnosis. *Influenza*—High fever, intense pain in the head, back, and limbs, and great prostration will serve to distinguish influenza from bronchitis when the former is prevalent.

Catarrhal Pneumonia.—Moderately high and irregular fever, prostration, dyspnœa, and physical signs indicating consolidation will serve in the recognition of pneumonia.

Prognosis.—Favorable. In the old, young, and feeble there is danger of its leading to capillary bronchitis or catarrhal pneumonia.

Treatment.—The abortive treatment consists in the use of hot foot-baths, a mustard plaster to the chest, the internal administration of hot drinks, and a full dose of Dover's powder (gr. x) with which quinine may be advantageously combined. This method is only applicable in the initial stage, and to those patients who are willing to remain indoors for the following twenty-four hours.

The young, old, and enfeebled should be confined to bed. A turpentine stupe, mustard plaster, or iodine may be applied to the chest.

In the early stage when there is substernal pain with little or no expectoration, sedative expectorants, like ipecac, the vegetable salts of potassium, antimony, and apomorphine are indicated; and it is well to combine with them an opiate to check the harassing cough.

℞ Potass. citrat., ℥ss;
Apomorphinæ hydrochlor., gr. j;
Syr. ipecac., f℥ss;
Succi limonis, f℥ij;
Syr. simp., q. s. ad f℥iv.—M. (Wood.)

Sig.—A dessertspoonful, in water, every three hours.

Or—

℞ Vini ipecacuanhæ, f℥ij;
Liq. potass. citrat., f℥iv;
Tinct. opii camph.,
Syr. acaciæ, āā f℥j.—M. (DaCosta.)

Sig.—Tablespoonful thrice daily.

In severe cases with dyspnœa, inhalations from a steam atomizer often give relief. Wine of ipecac (with twice its volume of water), tincture of lobelia, or tincture of conium may be employed for this purpose.

In the later stages, when expectoration has been established, stimulating expectorants are useful, such as ammonium chloride, squills, terpin hydrate, terebene, tar, or eucalyptus.

℞ Morphinæ sulphatis,
Potassii cyanidi, āā gr. iss;
Terpini hydratis, gr. xl;
Olei eucalypti, f℥j.

Pone in capsulas No. xx.

Sig.—One every two hours.

Or—

℞ Tinct. opii camph., f℥ij;
Syr. prun. virgin., f℥iss;
Syr. picis liquidæ, q. s. ad f℥iv.—M.

Sig.—A tablespoonful thrice daily.

Or—

℞ Terebeni, f℥ss.

Sig.—Five drops on sugar, gradually increased to ten thrice daily.

Chronic Bronchitis.

(Chronic Bronchial Catarrh, Winter Cough.)

Etiology.—It may result from the continuation of an acute attack; but more frequently it develops gradually in association with gout, alcoholism, or chronic heart or kidney disease. It is especially common in the old.

It is an associated condition in emphysema, phthisis, chronic interstitial pneumonia, and in many cases of asthma.

Pathology.—The mucous membrane of the bronchi is sometimes thickened and roughened from an overgrowth of the connective tissue; in other cases the mucosa is thin from atrophic changes. The surface is usually covered with mucopus; ulcers are occasionally noted.

Long-standing bronchitis leads to dilatation of the tubes (Bronchiectasis) and to emphysema.

Symptoms.—Persistent cough, and more or less muco-purulent expectoration; a sense of soreness behind the sternum. Fever is usually absent, and unless the disease is very severe, the general health may be fairly well preserved. Dyspnœa on exertion is a troublesome symptom; it however belongs more to the resulting emphysema than to the bronchitis.

Physical Signs.—Unless emphysema has developed, inspection, palpation, and percussion give negative results.

Auscultation reveals râles, some of which are dry and wheezing, while others are moist and bubbling.

Special Varieties.—(1) Rheumatic bronchitis. (2) Bronchorrhœa. (3) Dry catarrh.

Rheumatic Bronchitis.—This form occurs in those of a rheumatic diathesis, and is characterized by severe paroxysmal cough, the expectoration of scanty tenacious mucus, and by aching pains in various parts of the chest. It is especially influenced by atmospheric changes, and does not yield to the ordinary treatment of bronchitis.

Bronchorrhœa.—This term is applied to cases of chronic bronchitis which are associated with a very copious expectoration. The sputum is generally muco-purulent, and sometimes very offensive (Fetid bronchitis).

Dry Catarrh.—This form, described by Laennec as *catarrhe*

sec, is characterized by severe spells of coughing which are accompanied by little or no expectoration. It is generally seen in the old in association with emphysema or asthma.

DIAGNOSIS. *Phthisis.*—The absence of fever, of hemorrhage, of bacilli in the sputa, and of signs indicating consolidation will serve to distinguish chronic bronchitis from phthisis.

Bronchiectasis.—This often results from chronic bronchitis. Very profuse fetid sputa, expelled periodically in gushes, and perhaps physical signs of cavity over the main bronchi, posteriorly, indicate bronchiectasis.

Emphysema.—Much dyspnœa, distention of the chest, hyperresonance on percussion, and a prolonged feeble expiration on auscultation indicate emphysema.

SEQUELÆ.—Emphysema, bronchiectasis, and dilatation of the right ventricle

PROGNOSIS.—Perfect recovery is rarely attainable, but the disease is not incompatible with long life.

TREATMENT.—A careful regulation of the hygiene; this includes attention to diet, clothing, bathing, exercise, etc. Bronchitis dependent on heart or kidney disease will require remedies directed to those organs. The general vitality is frequently reduced, and tonics like cod-liver oil, hypophosphites, iron, quinine, and strychnine are often valuable adjuncts to the special treatment. A change of climate often secures permanent relief. In this country the extreme south-western territory, including New Mexico, Arizona, and Southern California, possesses many atmospheric advantages.

Alteratives like iodide of potassium (gr. v–x thrice daily) are often serviceable in chronic bronchitis with little expectoration.

Counter-irritants—blisters, tincture of iodine, or croton oil—prove useful.

Stimulating expectorants—chloride of ammonium, terebene, tar, eucalyptus, oil of sandalwood, and copaiba—are generally indicated :—

℞ Strychninæ sulphatis, gr. ss;
Codeinæ, gr. vj;
Terebeni,
Olei santali, āā f℥ss.

Pone in capsulas No. xii.

Sig.—One every three hours.

Or—

℞ Copaibæ, ʒiij;
Acaciæ et sacchar. alb., āā q. s.;
Spt. lavandulæ comp., fʒss;
Aquæ, q.s. ad fʒvj.—M.

Sig.—A tablespoonful thrice daily.

Or—

℞ Apomorphinæ hydrochlor., gr. ½;
Syr. prun. virg., fʒij;
Syr. picis liquidæ, fʒiv.—M. (Murrell.)

Sig.—A tablespoonful thrice daily.

The method of treating chronic bronchitis by inhalations, which has been so ably advocated by Dr. Murrell of London, is extremely useful, especially in patients with weak stomachs, in whom syrups should be avoided.

Wine of ipecac (with twice its volume of water), terebene (with equal parts of benzoinol or liquid vaseline), creosote, or carbolic acid may be so employed.

℞ Acid. carbol., gr. xxx;
Tinct. opii camph., fʒiij.—M. (N. S. Davis.)

Sig.—A fluid drachm with half a pint of hot water in the inhaler, thrice daily.

Fibrinous Bronchitis.

(Croupous Bronchitis, Pseudo-membranous Bronchitis.)

Definition.—A primary inflammatory disease of the bronchi associated with the formation of false membrane.

Etiology.—The causes are unknown. Male sex, early manhood, and chronic pulmonary disease, like phthisis, emphysema, and pleurisy, appear to be predisposing factors.

Pathology.—The disease is often limited to a certain number of bronchi. Some of the affected tubes are found filled with a fibrinous exudate, while others are found empty and show a loss of epithelium. The casts are usually expelled in the form of whitish balls, and when unrolled in water present branching moulds of the divisions and subdivisions of the affected bronchi. On close examination they are found to be hollow and laminated. Under the microscope, a homogeneous or fibrillated membrane is observed, imbedded in which are

leucocytes, fat-drops, particles of pigment, epithelial cells, and occasionally Leyden's octahedral crystals.

Symptoms.—Acute and chronic forms are recognized. The former is rare, and manifests the symptoms of a severe attaek of acute bronehitis, but the sputa contain fibrinous casts, and there is marked dyspnœa.

The ehronic form is charaeterized by severe cough, paroxysms of dyspnœa, and the expeetoration of fibrinous plugs. The physical signs are those of ehronic bronchitis. The disease often lasts a few weeks, and then disappears to return again at definite periods.

Prognosis.—In the acute variety the prognosis must be guarded; death frequently results from suffocation.

The chronic variety runs a very protracted course.

Treatment.—In the aeute disease, the atmosphere of the room should be kept moist and uniformly warm. Calomel (gr. $\frac{1}{4}$ every two hours) may be administered as in other membranous inflammations, and may be followed by iodide of potassium. Inhalations of alkaline vapors (lime-water) exert a solvent effeet. Counter-irritants should be applied to the ehest. Emetics sometimes aid in the expulsion of casts.

In the chronie form iodide of potassium may be given in conjunction with stimulating expectorants.

DILATATION OF THE BRONCHIAL TUBES.

(Bronchiectasis.)

Definition.—A universal or eireumscribed dilatation of the bronehi.

Etiology.—Chronic inflammation of the tubes and the eontraction of surrounding pulmonary tissue are the prime causes; hence, it is generally seeondary to chronic bronchitis, phthisis—particularly fibroid—chronic interstitial pneumonia, and ehronic pleurisy with adhesions.

Pathology.—The dilatation results from weakening and atony of the tubes, and from their subjection to strain in coughing, or to the traction of shrinking eonnective tissue, as in fibroid phthisis.

Two forms are noted: (1) The cylindrical form, in whieh

the tubes, particularly those of medium size, are uniformly dilated in one or both lungs; and (2) the saccular form, in which the tubes swell out, here and there, into circumscribed dilatations which may reach several inches in diameter. This form is especially noted in fibroid phthisis. The walls of the bronchiectatic cavity are extremely atrophied, the surface is generally smooth and shining, but ulcerations are not uncommon.

Symptoms.—Cough, dyspnœa, and copious expectoration. The last is characteristic; it is apt to occur periodically in gushes; the material has a highly offensive odor, and when allowed to stand in a glass vessel separates into three layers: an upper layer of dirty brown froth, a middle layer of turbid mucus, and an under layer of decomposed pus. Microscopically it contains pus corpuscles, fat crystals, crystals of hæmatoidin, and numerous microörganisms, but no tubercle bacilli. Elastic fibres are rarely found.

Physical Signs.—In the cylindrical variety the signs are those of chronic bronchitis. The saccular variety may present the signs of tuberculous cavities, localized tympany, cavernous breathing, gurgling râles, and pectoriloquy.

Diagnosis.—The differentiation of bronchiectasis from *phthisis* is difficult and often impossible. The discovery of tubercle bacilli always indicates phthisis. Bronchiectatic cavities are usually located in the lower lobes, and rarely in the apices.

Prognosis.—This will depend on the primary disease; since the common causes are long-standing bronchitis and fibroid phthisis, there can be little hope of cure. Amelioration is all that can be expected.

Treatment.—Tonics are often indicated. Stimulant and antiseptic expectorants like turpentine, terebene, eucalyptus, oil of sandalwood, and tar are sometimes useful.

Inhalations of terebene, carbolic acid, or dilute peroxide of hydrogen lessen cough and destroy the fetid odor of the breath. Codeine (gr. $\frac{1}{4}$) may be employed to allay cough.

ASTHMA.

Definition.—Paroxysmal dyspnœa due to spasm of the tubes or to swelling of their mucous membrane.

Etiology.—Asthma is a symptom of several diseases, but a hypersensitive condition of the mucous membrane of the respiratory tract appears to be essential to its production. When this condition prevails, asthma may be induced (1) by the pulmonary congestion of cardiac disease (Cardiac asthma); (2) by the uræmic intoxication or transient pulmonary œdema of Bright's disease (Renal asthma); or (3) by some irritant from without, as the pollen of plants (Hay asthma). (4) Sometimes the paroxysms are excited by the most trivial causes, as an atmospheric change or a peculiar odor, and to this form many writers restrict the term asthma. This last will be discussed under the head of essential asthma.

Essential Asthma.

(Bronchial Asthma, Nervous Asthma, Spasmodic Asthma.)

Etiology.—Nervous temperament, an hereditary tendency, early life, disease of the naso-pharynx, and the gouty diathesis, are predisposing factors.

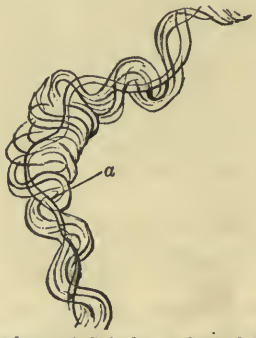
Barometric and thermometric changes; the inhalation of dust; the odor of certain plants, animals, or fruits; excitement; reflex irritation, particularly a loaded stomach; a change of locality; and bronchial catarrh, are exciting causes.

Pathology.—The disease is a pure neurosis, and the paroxysms probably result from a spasm of the smaller tubes, or turgescence of their mucous membrane.

Symptoms.—The paroxysms often appear suddenly, but in some cases certain symptoms precede and give warning of the approaching attack; among these are chilliness, flatulence, sneezing, and a copious discharge of pale urine. The patient is often seized at night. There is a sense of oppression and anxiety followed by dyspnœa so intense that he runs to the window for air, or sits upright with his arms in such a position that he can bring into play the auxiliary muscles of respiration. The face is pale, the lips blue, the eyes prominent and con-

gested, and the body cold and covered with sweat. The respirations are not rapid, but labored and noisy. Cough is often present and is associated with the expectoration of scanty viscid mucus. On close examination little grayish balls are noted in the sputum, and when unravelled, they are found to be composed of delicate spirals of mucus, which have been moulded in the finer bronchioles (Curschmann's spirals).

Fig. 16



Curschmann's Spirals. *a*, Central fibre.

Microscopic examination also reveals octahedral crystals similar to those found in leukæmia (Charcot-Leyden crystals).

The paroxysms may last from a few minutes to many hours, and may recur for several successive nights, or may disappear entirely for weeks or months.

PHYSICAL SIGNS.—*Inspection* reveals evidences of dyspnœa and distention of the chest.

Percussion generally yields hyper-resonance.

Auscultation.—A prolonged, high-pitched, wheezing expiration, with abundant sonorous and sibilant râles. The expiratory wheezing may be audible over the entire room.

DIAGNOSIS.—*Cardiac and renal asthma* are to be distinguished from essential asthma by the history, and by the evidence of organic heart or kidney disease.

Hay asthma is recognized by the associated coryza and by its periodic occurrence every spring or fall.

Laryngeal obstruction from foreign bodies, croup, paralysis of the vocal cords, or œdema.—The dyspnœa is with inspiration, and the chest instead of being distended is retracted, especially at the base.

Sequelæ. — Emphysema invariably follows when the asthma is of long duration; it results from the tension to which the vesicles are subjected during the expiratory effort. Dilatation of the right ventricle is also a remote sequel.

Prognosis. — The disease does not prove fatal except through complications or sequelæ. In young persons without an inherited tendency the prognosis should be guardedly favorable; it frequently subsides at puberty. Cases associated with some definite reflex cause, as nasal obstruction, often recover when the latter is removed. The older the patient, the greater the inherited tendency, the more unfavorable becomes the prognosis.

Treatment. *The Attack.*—Prompt relief often follows the inhalation of nitrite of amyl (five or six drops in a glass or on the handkerchief), iodide of ethyl (twenty to thirty drops), or a few whiffs of chloroform. Smoking cigarettes of belladonna and stramonium leaves wrapped in nitre-paper—paper which has been soaked in a saturated solution of saltpetre and dried—will often suffice in mild attacks. Nitre-paper may be burned in the room and the fumes inhaled.

The application of dry cups or thin poultices to the chest is often a valuable adjunct to the treatment. Morphine (gr. $\frac{1}{8}$–$\frac{1}{4}$) with sulphate of atropine (gr. $\frac{1}{120}$) will often cut short an attack. *Internally*, sedatives like Hoffmann's anodyne (ʒss), tincture of lobelia (♏ xx), and bromide of potassium (gr. xxx), are sometimes useful.

℞ Tinct. belladonnæ,
Tinct. lobeliæ, āā fʒiiss;
Spiritus æther. comp.,
Tinct. opii camph., āā fʒvj;
Syrup. prun. Virginianæ, q. s. ad fʒiv.—M.
Sig.—A dessertspoonful every three hours.

The Interval.—Careful search should be made for some reflex irritation, especially in connection with the naso-pharynx. An easily-assimilable diet must be selected; in nocturnal

asthma the evening meal should be very light. Graduated exercise and frequent bathing, followed by friction of the skin, will add to the general vigor. A change of climate is desirable, but there is no fixed rule in the selection of locality. Many asthmatics do well in the city, but a dry atmosphere and a high altitude are better suited to the majority. Busey claims excellent results from the habitual wearing of an oil-silk jacket in asthma associated with bronchitis. Among the remedies arsenic and iodide of potassium hold a high place as alteratives. Fowler's solution (three drops, gradually increased to ten or more, thrice daily), or five to ten grains of the iodide may be administered over long periods. Nitroglycerin (gr. $\frac{1}{100}$), or nitrite of sodium (gr. iij–v thrice daily) often gives immunity for long periods.

HAY ASTHMA.

(Hay Fever, Autumnal Catarrh, Rose Cold.)

Definition.—A catarrhal affection of the respiratory tract, usually occurring periodically every spring or autumn, excited by the action of some atmospheric irritant upon a hyperæsthetic mucous membrane, and characterized by coryza, bronchitis, and asthmatic seizures.

Etiology.—An inherited tendency, male sex, nervous temperament, indoor life, and chronic nasal catarrh are predisposing factors. The attack as a rule occurs in the autumn (Autumnal catarrh), or in the spring (Rose cold), and is excited by certain dusts, vapors, or odors. The pollen of plants seems to be a common excitant. The seizures may occur at any time if the peculiar irritant is present.

Pathology.—An essential feature is the hypersensitive condition of the mucous membrane, and this is often, though not invariably, associated with hypertrophic rhinitis.

Symptoms.—Redness of the conjunctivæ and swelling of the eyelids; pruritus of the pharynx, nose, and eyes; sneezing; obstruction of the nostrils; watering of the eyes; a copious discharge of mucus from the nose; headache; cough; and asthmatic attacks are the usual phenomena.

Rose cold usually begins in May or June and runs to the

latter part of July. *Autumnal catarrh* begins in the latter part of August and ends with the first frost.

PROGNOSIS.—The disease runs an indefinite course, and rarely, if ever, proves fatal. Cases which are associated with chronic rhinitis often permanently recover on the removal of the latter. In other cases, the prognosis as regards immunity from future attacks is unfavorable.

TREATMENT.—Careful search should be made for chronic nasal disease, and if found, appropriate treatment instituted.

A change of climate during the period of susceptibility exempts most patients. A sea-voyage or a sojourn in some high-mountain district, like the White Mountains, Adirondacks, Catskills, or Alleghanies may be recommended.

Tonics are usually indicated, and quinine, arsenic, and strychnine are often very useful when administered before and during an attack. To allay itching and lachrymation, the eyes may be washed with a solution of boric acid (gr. x to ℥j), or sulphate of zinc (gr. i–ij to ℥j). Sneezing, nasal fulness, and discharge are often relieved by medicated sprays. A solution of cocaine, or the following may be employed :—

℞ Menthol, ʒj–ʒij ;
Ol. amygd. dulc. *vel* benzoinol, f℥ij–M.
Sig.—Spray into the nose and throat every few hours.

PULMONARY EMPHYSEMA.

DEFINITION.—Abnormal distention of the lungs with air.

VARIETIES.—(1) Interlobular emphysema: This form is rare, and results from the rupture of the lung and escape of air into the interstitial tissue. (2) Compensatory emphysema: When a lung or a part of a lung is disabled from any cause, the healthy portions distend and do vicarious work. (3) Atrophic or senile emphysema: In old people the solids of the lung atrophy, so that a relative increase of air results. (4) Hypertrophic emphysema. The last three varieties are included under the term vesicular emphysema.

Hypertrophic Emphysema.

Definition.—A pulmonary disease characterized anatomically by dilatation of the air-vesicles and atrophy of their walls; and clinically by dyspnœa, enlargement of the thorax, hyper-resonance, and weak breathing.

Etiology.—Congenital weakness of the lung structure—probably a defective development of elastic tissue—is an important predisposing factor. This predisposition may be transmitted through several generations.

In forced expiration, the air cannot escape with sufficient rapidity through the narrow glottis, and the backward pressure stretches the air-vesicles; hence, the obstinate cough of chronic bronchitis, the expiratory straining of asthma, and occupations which necessitate forced expiration, like playing on wind instruments and glass-blowing, are causal factors.

Pathology.—The lungs are enlarged, and do not collapse when the thorax is opened. In bad cases the free margins are studded with large bullæ or blebs which have resulted from the rupture of a number of vesicles into a common sac. The organs are pale, and have a soft cotton-like feel. Microscopic examination reveals atrophy of the vesicular walls, a diminished amount of elastic tissue, and more or less obliteration of the pulmonary capillaries. This last condition leads to increased tension in the pulmonary artery and to secondary hypertrophy of the right ventricle.

Symptoms.—The disease generally manifests itself in middle life, but it is not infrequently observed in the young. Dyspnœa, increased by exertion; cyanosis, often extreme during attacks of acute bronchitis; and cough, from the associated bronchitis, are the usual symptoms. In advanced cases dropsy may result from cardiac failure.

Physical Signs.—The neck is short, and the sterno-cleido-mastoids prominent. The thorax is likewise short, but broad especially in its antero-posterior diameter. This configuration has given rise to the term "barrel-shaped" chest. On respiration there is little expansion, but an elevation of the thorax as a whole. The apex-beat is invisible, but an abnormal pulsation is often noted in the epigastrium.

Palpation.—Diminished vocal fremitus.

Percussion.—Increased resonance. The upper level of hepatic dulness is depressed, and the area of cardiac dulness may be almost obliterated.

Auscultation.—Inspiration is short, expiration is prolonged and low-pitched, or inaudible. Râles resulting from the associated bronchitis are frequently heard. The pulmonary second sound is accentuated.

Complications.—Bronchitis, asthma, dilatation of the right ventricle, and later, tricuspid regurgitation and dropsy.

Diagnosis. *Chronic Bronchitis.*—The dyspnœa, thoracic enlargement, hyper-resonance, and prolonged expiration separate emphysema from bronchitis.

Pneumothorax.—This is almost invariably unilateral, the resonance is tympanitic, and metallic tinkling and bell-tympany are obtained on auscultation.

Prognosis.—The disease is generally incurable; but its advance may be stayed by relieving the primary condition. Emphysema runs a long course and is in itself rarely fatal, but death may result from heart failure and dropsy, or from intercurrent pneumonia.

Treatment.—The remedies advocated in chronic bronchitis and asthma are often applicable here. The patient should be placed under the most favorable hygienic conditions. Iodide of potassium (gr. x thrice daily) is often used empirically, and sometimes relieves the dyspnœa and cough. Iron is indicated in the anæmie. Strychnine (gr. $\frac{1}{40}$—$\frac{1}{30}$) is a valuable respiratory and cardiac stimulant, and may be combined with digitalis when there are symptoms of heart failure.

℞ Strychnin. sulph., gr. ½;
Pulv. digitalis,
Pulv. scillæ,
Ferri reduct., āā gr. xx.—M.
Ft. in pil. No. xx.
Sig.—One thrice daily.

The inhalation of oxygen, or the inspiration of compressed air followed by expiration into rarefied air is sometimes a useful measure.

HÆMOPTYSIS.

(Bronchorrhagia, Broncho-pulmonary Hemorrhage.)

DEFINITION.—The expectoration of blood.

ETIOLOGY.—(1) Vicarious menstruation (rare). (2) Traumatism. (3) Inflammatory diseases of the respiratory tract, especially phthisis and pneumonia. (4) The rupture of an aortic aneurism. (5) Obstruction to the venous circulation as in chronic heart and liver disease. (6) Malignant disease of the lung. (7) A dyscrasia of the blood, as in purpura, the infectious fevers, hæmophilia (bleeder's disease), and scurvy. (8) It occasionally occurs in young people without obvious cause.

SYMPTOMS.—Sometimes the bleeding is preceded by cough, dyspnœa, or substernal warmth or tenderness, but often there is no premonition, and the first indication is the presence of a warm salty fluid in the mouth. The blood is generally raised by coughing, and is bright red and frothy. It is alkaline in reaction, and intimately mixed with air and mucus. The hemorrhage is rarely profuse unless it results from the rupture of an aortic aneurism or the ulceration of a large vessel in advanced phthisis. Auscultation of the chest reveals bubbling râles. The subsequent expectorations are tinged with blood, and if much is swallowed it may excite vomiting or pass into the intestine and impart a tarry appearance to the stools.

DIAGNOSIS.—*Hœmoptysis* must be distinguished from *hœmatemesis*:—

HÆMOPTYSIS.	HÆMATEMESIS.
History of some chest disease.	History of some abdominal disease.
The blood is ejected by coughing.	The blood is ejected by vomiting.
The blood is bright red and frothy.	The blood is dark, and dense or clotted.
The blood is mixed with sputum.	The blood is mixed with food.
The blood is alkaline in reaction.	The blood is acid in reaction.
The subsequent expectorations are tinged with blood, and the stools are rarely tarry.	The subsequent expectorations contain no blood, and the stools are frequently tarry.
Auscultation reveals râles.	Auscultation gives negative results.

Prognosis.—Hæmoptysis is rarely the cause of death in the disease in which it occurs. In phthisis the symptoms often improve after a moderate hemorrhage. On the other hand, in aneurism, advanced phthisis, and abscess and gangrene of the lung, the bleeding may prove fatal.

Treatment.—Absolute rest and the avoidance of excitement. The shoulders should be elevated; an ice-bag may be placed on the chest, and pieces of ice may be held in the mouth. and slowly swallowed. Morphine is generally required as a sedative; it may be given hypodermically with ergotin (gr. v–x) or with the fluid extract of ergot (♏ x–xx). Gallic acid (gr. x–xx) may be given by the mouth. Astringent sprays are useless. A saline purge may act beneficially by inviting blood away from the congested organ. A firm ligature around one or both legs retards the flow of venous blood, and so aids in arresting the hemorrhage.

When the bleeding is not profuse, but frequently repeated, the following internal remedies are efficient: Acetate of lead gr. ij with powdered opium gr. ¼, gallic acid (gr. x–xx), fluid extract of hamamelis (ʒj–ʒiij), turpentine (gtt. x), or—

℞ Acid. gallic., ʒiiss;
Acid. sulph. aromat., fʒj;
Glycerin., fʒss;
Aquæ, q. s. ad fʒiv—M.

Sig.—A tablespoonful thrice daily.

PULMONARY APOPLEXY.

(Hemorrhagic Infarction of the Lung.)

Definition.—An effusion of blood into the pulmonary tissues.

Etiology.—It may result from degeneration of the pulmonary vessels, but it is most frequently due to an embolus or a thrombus in one of the branches of the pulmonary artery. The embolus is usually a portion of a thrombus which has formed in the heart or in one of the systemic veins. Occlusion of the vessel causes a backward flow of blood, the part becomes engorged, and effusion follows.

PATHOLOGY. — The infarction is usually located in the periphery of the lung; it is conical in shape with its apex pointing inwards. The portion affected is airless, and reveals an infiltration of dark blood. Microscopic examination shows a dense aggregation of blood-corpuscles.

If it does not prove fatal, absorption and subsequent fibroid induration result.

SYMPTOMS.—When the infarction is large the usual symptoms are dyspnœa, cough, and the expectoration of dark blood containing few air-bubbles. These symptoms occurring in chronic heart-disease are especially suggestive.

Physical Signs.—Very large infarctions give dulness and bronchial breathing.

TREATMENT.—The condition itself is not amenable to treatment. Remedies should be directed to the primary disease.

CONGESTION OF THE LUNGS.

Active Congestion.

ETIOLOGY.—This results from increased afflux of blood to the lungs. Hypertrophy of the heart, violent exercise, mountain-climbing, the inhalation of irritants, and mental excitement occasionally produce it. It is an associated condition in all severe inflammatory diseases of the lungs. In the vast majority of cases it marks the initial stage of croupous pneumonia.

PATHOLOGY.—The lung is bright red in color, heavy, and less crepitant. When incised and pressed, copious frothy blood exudes.

SYMPTOMS. — Flushed face; dyspnœa; short, dry cough, followed by tenacious blood-streaked expectoration; and a rapid, full pulse. *Physical examination* reveals slight dulness, crepitant râles, and broncho-vesicular breathing.

TREATMENT.—Rest; liquid diet; wet cups to the chest.

Internally.—Veratrum viride and a saline purge.

Passive Congestion.

Etiology.—This results from obstruction to the flow of blood from the lungs to the heart. The chief cause is cardiac disease, especially fatty degeneration, dilatation, and mitral disease.

Pathology.—The lungs are dark red in color, and often somewhat œdematous. When the condition has lasted a long time, the organs become brown, dense, and tough (brown induration). Microscopic examination reveals a dilatation of the capillaries, an overgrowth of connective tissue, free pigment granules, and degenerative changes in the bloodvessels.

Symptoms.—Dyspnœa; hard cough; mucous expectoration containing pigmented cells. *Physical examination* reveals râles, slight dulness, and feeble breathing.

Treatment.—Remedies should be directed to the underlying cardiac disease. The application of dry cups often gives temporary relief. Saline laxatives may prove useful.

Hypostatic Congestion.

(Hypostatic Pneumonia, Splenization of the Lung.)

Definition.—A congestion of dependent portions of the lungs occurring in asthenic diseases which necessitate a protracted recumbent position.

Etiology.—It is generally observed in low fevers and in chronic wasting diseases. (1) Blood-dyscrasia, (2) a weak heart, and (3) a recumbent position are the causal factors.

Pathology.—The lungs are dark red and œdematous posteriorly. The œdema and increased amount of blood render the organs more solid and less crepitant. They never show the granular appearance of croupous pneumonia.

Symptoms.—Dyspnœa, cough, and scanty expectoration.

Physical examination reveals slight dulness, subcrepitant râles, and feeble bronchial breathing.

Treatment.—Efforts should be made to prevent the development of hypostatic pneumonia in asthenic disease by frequent change of position, and the timely use of such cardiac

stimulants as alcohol, strychnine, digitalis, ammonia, and turpentine. When already present, turpentine stupes or dry cups may be applied externally, and one or more of the above stimulants administered internally.

CROUPOUS PNEUMONIA.

(Lobar Pneumonia, Pneumonitis, Lung Fever.)

DEFINITION.—An acute specific disease, characterized anatomically by an inflammation of the lungs, followed by a rapid infiltration of their alveoli; and manifested clinically by high fever, cough, dyspnœa, "rusty" sputum, and physical signs indicative of consolidation.

ETIOLOGY.—Age, sex, and climate exert but little predisposing influence. Lowered vitality from bad hygiene or from some pre-existent disease, like diabetes, Bright's disease, or one of the infectious fevers, favors its development. One attack renders the patient more liable to subsequent infection. Alcoholism is a strong predisposing factor. Exposure to cold and wet often precipitates the attack.

The exciting cause is the invasion of the lung by pathogenic bacteria, especially by Fränkel's diplococcus pneumoniæ.

PATHOLOGY.—Anatomically three stages have been recognized: (1) The stage of congestion; (2) of red hepatization; (3) of gray hepatization.

Stage 1.—The affected portion remains distended when the chest is opened; it is of a deep-red color, and is more resistant to the touch than the normal lung. On section, a frothy blood-stained serum freely exudes. Microscopic examination reveals a dilated and tortuous condition of the capillaries, swelling of the alveolar cells, and a slight corpuscular exudate.

Stage 2.—The hepatized portion is increased in volume, is quite firm, is of a dark-red color, and so heavy that it sinks in water. It is very friable, and the torn surface presents a granular appearance from the projection of the fibrinous plugs in the alveoli.

Microscopic examination reveals a mesh of coagulated fibrin, enclosing numerous red blood-corpuscles and some leucocytes;

the latter are also noted in the interlobular tissue. In sections properly treated the diplococcus is detected.

Stage 3.—The red color gives place to a mottled gray, and the solidified lung begins to soften. The change in color is due to the compression of the capillaries, to the disappearance of red corpuscles and their replacement by leucocytes, and to fatty degeneration of some of the elements.

In favorable cases resolution occurs before gray hepatization has far advanced, the exudation being removed by absorption and expectoration.

In unfavorable cases the consolidated lung may become infiltrated with pus (Purulent infiltration); it may become gangrenous; or, very rarely, it may become the seat of fibroid induration (Chronic interstitial pneumonia).

Death may result early in the disease from the generated blood-poisons, or from rapid diminution of the respiratory surface.

The consolidation usually begins at the base and extends upwards. The most frequent seat is the lower lobe of the right lung. The bronchi and the adjacent pleura are involved in the inflammatory process.

Symptoms.—The disease usually begins with a decided chill and a sharp pain in the side, followed by a rapid rise of temperature; the latter often attains its maximum (104°–105°) in twenty-four hours, and generally continues high, with slight diurnal remissions, until the ninth day, when it falls by crisis, frequently reaching the norm by the tenth day. Occasionally the temperature falls by lysis. There is marked dyspnœa; the respirations are shallow and rapid, ranging from 40 to 80 per minute, thus making the ratio between respiration and the pulse 1 to 3 or 1 to 2. Cough is a prominent symptom; at first it is short and dry, but later it is accompanied by bloody ("rusty"), translucent, and tenacious sputa. Microscopically the sputum contains red blood-corpuscles, their free pigment, pus-corpuscles, diplococci, and other microörganisms. The face is flushed; the lips are cyanosed and often the seat of an herpetic eruption; the tongue is heavily furred; the bowels are constipated; and the urine is scanty, high-colored, deficient in chlorides, and often slightly albuminous. In severe

cases delirium is rarely absent. Examination of the blood usually shows marked leucocytosis.

PHYSICAL SIGNS. *Inspection.*—Diminished expansion, but no bulging of the interspaces or displacement of the apex-beat.

Palpation. — Diminished expansion and increased vocal fremitus.

Percussion.—At the onset there may be tympany over the affected area from diminished intra-pulmonary tension. As consolidation advances the note becomes remarkably dull. Exaggerated resonance is noted around the hepatized areas.

Auscultation.—In the stage of congestion fine crepitant râles are heard at the end of forced inspiration; they probably result from the forcible separation of adherent vesicular walls, and disappear when the lung becomes solidified. Auscultation then detects increased vocal resonance, and harsh breathing which is prolonged, high-pitched, and tubular in expiration (bronchial).

During resolution the softened exudate produces fine moist râles—the redux-crepitus.

Atypical Cases. *Senile Pneumonia.*—The symptoms often develop insidiously; the temperature may not be high; the pulse may not be accelerated; expectoration is often absent; the signs are not marked; delirium is common; weakness is extreme; and death from exhaustion is the most frequent termination.

Pneumonia in Children.—It is often ushered in with convulsions. Headache, delirium, stupor, and coma are prominent symptoms, so that the disease may simulate meningitis. The temperature is very high; expectoration is often absent. The disease frequently begins at the apex of the lung.

Typhoid Pneumonia.—Pneumonia associated with typhoid symptoms, — headache, muttering delirium, stupor, a dry, brown tongue, subsultus tendinum, carphologia, a rapid, weak pulse, and high fever which, in favorable cases, falls by lysis. The expectoration is often like prune-juice.

Pneumonia of Drunkards.—The onset is gradual; the expectoration is like prune-juice; the temperature is not high, but a violent maniacal delirium commonly develops and is followed by death from exhaustion.

Complications.—Pleurisy, pericarditis, malignant endocarditis, œdema of the lungs, delayed resolution (consolidation may last five or six weeks, and then disappear), abscess of the lung, gangrene of the lung, and chronic interstitial pneumonia.

Diagnosis. *Pleurisy.*—Here the initial chill is not so marked; the fever is not so high nor the pulse so rapid; and there is no "rusty" sputum; but bulging and displacement of the apex-beat are often noted on inspection; the percussion-dulness may change with the posture of the patient; vocal resonance and vocal fremitus are diminished; and the breathing is distant and weak.

Acute Phthisis.—Irregular fever, bacillus tuberculosis in the sputum, and the continuation of grave symptoms with signs of softening after the ninth or tenth day, will suggest the diagnosis of tuberculosis.

Pulmonary Œdema.—Here there is absence of chill, fever, and pain; the expectoration is watery, not "rusty;" both lungs are commonly affected; auscultation reveals abundant subcrepitant râles and weak breathing.

Typhoid Fever.—Typhoid pneumonia may be readily mistaken for typhoid fever with pneumonia; but pneumonia as a complication occurs late in the disease, so that the history of the onset gives much assistance.

Prognosis.—In patients previously healthy the prognosis is good. At the extremes of life the outlook is grave. In drunkards the disease is especially fatal.

In individual cases, great dyspnœa and cyanosis, rapidly increasing consolidation, involvement of both lungs, muttering delirium, a failing pulse, the absence of leucocytosis, and a dark sputum are unfavorable factors.

The average mortality is 20 per cent.

Treatment.—Absolute rest. A liquid or semi-liquid diet (milk, koumiss, eggs, broths, beef juice). The chest should be enveloped in a cotton jacket covered with oiled silk.

Although pneumonia is an infectious disease which produces widespread disturbance in the economy, the immediate danger is often obstruction to the pulmonary circulation; so that in the stage of congestion, when the pulse is full and strong, veratrum viride (℥ iij–v of the fluid extract every hour until

the pulse softens) is a valuable remedy. It depresses the heart, dilates the systemic vessels, and so invites blood away from the engorged lung. In the very robust, venesection may be substituted for veratrum.

In consolidation, the right ventricle is subjected to a strain and there is danger of heart failure; hence cardiac stimulants are indicated in this stage. The tincture of digitalis (gtt. x every two or three hours, being guided by the pulse) may be given by the mouth; when the stomach is irritable, the drug should be administered hypodermically. Strychnine (gr. $\frac{1}{30}$) is also of great value as a cardiac and respiratory stimulant. Ammonia is useful in some cases, and either the aromatic spirits or the carbonate may be employed. The inhalation of oxygen sometimes gives much relief. Marked cyanosis with engorgement of the right ventricle is an indication for venesection.

As a general stimulant and food, alcohol is often indicated. In typhoid pneumonia turpentine (℥ v) may be associated with the alcohol.

Pain may be relieved by opium, or by the application of wet cups, dry cups, an ice-bag, or hot fomentations.

Delirium.—Apply an ice-bag to the head, and administer bromide of potassium, hyoscine, musk, or camphor internally. When the delirium is associated with high fever, a cold pack or tepid bath will often control it.

Pyrexia.—Occasionally, high fever will require treatment; sponging, a cold pack, or a cold bath (80°) may be employed. Antipyrin (gr. vj) is a safe and efficient remedy.

Convalescence should be guarded, and such tonics as iron, quinine, strychnine, and cod-liver oil will be found useful restoratives.

In delayed resolution, small blisters may be applied over the affected areas, and iodide of potassium may be administered internally. Thus:—

Potass. iodid., ʒj;
Ammon. chlor., ʒiss;
Mist. glycyrrhizæ comp., fʒvj.—M. (Da Costa.)
Sig.—Tablespoonful four times a day.

CATARRHAL PNEUMONIA.

(Broncho-pneumonia, Lobular Pneumonia, Insular Pneumonia.)

DEFINITION.—An inflammation of the terminal bronchioles and air-vesicles.

ETIOLOGY.—It is most frequently observed in the very young and the old. It is a common sequel of the specific fevers, especially of whooping-cough, measles, influenza, and diphtheria. In debilitated subjects it may occur as a primary affection, the result of exposure.

Another group of cases results from the aspiration of particles of food into the smaller bronchi (aspiration or deglutition pneumonia). This accident is liable to occur whenever the sensibility of the larynx is benumbed, as in apoplexy, bulbar palsy, or uræmia. Cancer of the throat and operations on the upper air-passages also favor its occurrence. The pathogenic bacteria are the pneumococci, the streptococci and staphylococci of suppuration, and the bacillus of influenza.

PATHOLOGY.—As a rule, both lungs are involved. On section, small projecting areas of consolidation are noted here and there around the finer bronchioles. Recent patches are reddish-brown in color, firm, and smooth or finely granular; later they become grayish and soft. The terminal bronchi are filled with purulent material.

In addition to these solidified areas, there are other small patches of collapsed lung which are airless, firm, and bluish-red in color. The collapse has resulted from occlusion of the bronchus, and closely resembles consolidation; but it can, as a rule, be overcome when inflation is practised by means of a blowpipe inserted in the supplying bronchus.

Microscopic examination reveals an exudate in the terminal bronchi and air-cells, which is composed of leucocytes and desquamated epithelium in various stages of degeneration. The walls of the bronchi are also infiltrated with leucocytes.

When compared with croupous pneumonia, the contrast is striking. In the latter the lung is involved *en masse;* the consolidation is distinctly granular, and is composed of red blood-

corpuseles, white blood-corpuseles, fibrin, and diplococci; the lining epithelium is but slightly involved; and the walls of the bronchi are not infiltrated with leucocytes.

Terminations.—(1) Resolution; the exudate undergoes fatty degeneration and is removed by absorption or expectoration. (2) Tuberculosis. Termination in phthisis is quite common; doubtless in many cases the disease was primarily tuberculosis, and in others the exudate became a good soil for the development of tubercle bacilli. (3) Abscess or gangrene; these terminations are rare except in pneumonias resulting from aspiration.

Symptoms.—The symptoms are often masked by the primary disease. The onset is usually gradual, and is characterized by prostration, cough, and fever. The last is moderately high and very irregular (101°–104°). The dyspnœa is marked, and the respirations are rapid—50 to 80 per minute; the pulse is greatly accelerated—120 to 180 per minute; cough is painful and accompanied by a mucо-purulent expectoration which is rarely blood-streaked. The face is usually pale and anxious, and the lips blue.

Physical Signs.—As the areas of consolidation are generally small and scattered, the physical signs are not marked.

Inspection reveals evidences of dyspnœa,—lividity, playing of the nostrils, prominence of the sterno-cleido-mastoids, and retraction of the base of the chest.

Palpation usually gives negative results.

Percussion may reveal areas of dulness in one or both lungs.

Auscultation reveals fine sibilant (whistling) or subcrepitant râles, and areas over which the breathing is tubular, or bronchial.

Diagnosis.—The following table will show the clinical differences between *catarrhal* and *croupous pneumonias*:—

	Catarrhal Pneumonia.	Croupous Pneumonia.
Cause	Usually secondary to bronchitis.	A primary disease excited by the diplococcus.
Onset	Gradual, a chill generally absent.	Abrupt onset with a chill.
Fever	Moderately high, very irregular, and ending by lysis after an indefinite period.	High, regular, and ending by crisis at the eighth or ninth day.
Expectoration .	Muco-purulent.	"Rusty," translucent, and tenacious.
Physical Signs .	A bilateral disease. Physical signs are indistinct and indicate scattered areas of consolidation.	A unilateral disease. Physical signs are distinct and indicate a large and uniform consolidation.

Acute Phthisis.—In this disease there is a tuberculous broncho-pneumonia which is difficult to distinguish from simple broncho-pneumonia. A family history of tuberculosis, an extensive involvement of the apices, bubbling râles indicating softening, long duration, and bacilli and elastic fibres in the sputa are the diagnostic phenomena of phthisis.

Bronchitis.—In simple bronchitis the fever is not high, the dyspnœa is rarely marked, prostration is usually absent, and there are no physical signs indicating consolidation.

Prognosis.—Always guarded. In the very young, very old, and debilitated the disease is commonly fatal. Many recover from the pneumonia following the infectious fevers. Aspiration-pneumonia is commonly fatal. The mortality is difficult to estimate, for acute phthisis is often diagnosed catarrhal pneumonia; it is probably greater than in croupous pneumonia, and varies from 30 to 60 per cent. The duration is from one to three weeks; a longer duration would suggest tuberculosis.

Treatment.—The disease can often be prevented by carefully protecting patients suffering from bronchitis and infectious fevers. In the latter it is also essential that the nasopharynx should be kept clean with some mild antiseptic solution.

The room should be well ventilated, but free from draft,

and the temperature should be kept uniformly at 70°. A moist atmosphere is desirable, and an apparatus for producing steam may be improvised. Tincture of iodine may be applied locally, and the chest enveloped in a cotton jacket.

The diet should be liquid or semi-liquid, and may include milk, junket, koumiss, eggs, broths, and beef-juice. Stimulants, wine or brandy, are usually required to combat the extreme prostration.

At the onset a laxative should be administered, and calomel may be selected (gr. $\frac{1}{8}$ every hour until it operates).

Stimulating expectorants are nearly always indicated, and chloride of ammonium, carbonate of ammonium, squills, or senega may be employed.

℞ Ammon. chloridi, gr. 1;
Spt. ætheris nitrosi, f℥ss;
Syr. senegæ, f℥iiss;
Tinct. cardamom. comp. et aquæ, āā q. s. ad f℥ij.
—M.

Sig.—A teaspoonful every two or three hours to a child of three years.

Or—

℞ Ammon. carb., gr. xxiv;
Syr. tolu., f℥vj;
Spt. vini gal., f℥iij;
Syr. senegæ, f℥iijss;
Syr. acaciæ, q. s. ad f℥iij.—M.
(Goodhart and Starr.)

Sig.—Teaspoonful every two hours to a child of two or three years.

Strychnine is often invaluable as a respiratory and cardiac stimulant; for an adult, gr. $\frac{1}{30}$ may be given three or four times daily.

The accumulation of mucus in the bronchial tubes, indicated by extreme cyanosis, a weak pulse, and bubbling râles, will call for an emetic; wine of ipecac (℥j–℥ss), or apomorphine (for an adult gr. $\frac{1}{12}$) may be selected. Nervous symptoms—restlessness, delirium, etc.—will often be relieved by a cold pack or by a cold bath. Hyoscine, bromide of potassium, or

chloral in small doses may be required. In children the following suppository is often very efficient :—

℞ Pulv. asafœtidæ, ʒj;
Quininæ sulph., gr. xxx;
Ol. theobromatis, q.s.—M. (Pepper.)
Ft. in suppos. No. xii. (Child's size.)
Sig.—One every three or four hours for a child of five years.

In delayed resolution counter-irritants should be applied to the affected areas, and iodide of potassium should be administered internally.

Convalescence must be guarded; tonics like cod-liver-oil, iron, arsenic, and hypophosphites are useful restoratives. A change of scene is desirable.

CHRONIC INTERSTITIAL PNEUMONIA.

(Cirrhosis of the Lung, Chronic Pneumonia, Pulmonary Induration.)

Definition.—A chronic disease of the lung, characterized by an overgrowth of fibrous tissue.

Etiology.—It is a rare sequel of croupous pneumonia. It is commonly found associated with tubercles in fibroid phthisis. The overgrowth of connective tissue is sometimes induced by an old fibrinous pleurisy. It may be an expression of syphilis. It arises primarily from the constant inhalation of irritating dusts, as stone-dust (Chalicosis), coal-dust (Anthracosis), metal-dust (Siderosis).

Pathology.—When the thorax is opened the lung is found retracted and the heart displaced. The organ is tough, firm, and more or less airless. Section shows an overgrowth of fibrous tissue, and usually inflammation and considerable dilatation of the bronchi.

Symptoms.—Moderate dyspnœa and chronic cough; the expectoration may be slight, but often it is profuse, and fetid from having been retained in bronchiectatic cavities. There is no fever, and the general health may be well preserved for many years.

Physical Signs. — *Inspection* reveals retraction of the affected side and displacement of the apex-beat.

Percussion often yields dulness; but over saccular dilatations of the bronchi there may be hyper-resonance.

Auscultation. — The vocal resonance is increased and the breathing is often bronchial or cavernous.

Diagnosis. *Fibroid Phthisis.*—Involvement of both lungs, bacilli in the sputa, and fever would indicate fibroid phthisis.

Prognosis.—Incurable. The duration is from ten to twenty years.

Treatment. — Palliative. It consists in good hygienic regulations and the use of remedies directed to the bronchiectasis.

GANGRENE OF THE LUNG.

Definition.—A putrefactive necrosis of the lung.

Etiology.—Gangrene is not a primary condition, but is secondary to some inflammatory disease of the lung. It is excited by the entrance of bacteria of putrefaction, but unless the system is considerably reduced in vitality the tissues, even though diseased, show wonderful resistance, and escape putrefaction.

Pneumonia, especially aspiration-pneumonia, phthisis, pressure of morbid growths, bronchiectasis, abscess, and hemorrhagic infarction following embolism of the pulmonary artery are the predisposing pulmonary conditions; and Bright's disease, alcoholism, the infectious fevers, and particularly diabetes, by lowering the vitality, render these conditions operative.

Pathology.—The process may be circumscribed or diffuse, most frequently the former. The affected part is converted into a greenish-black, soft mass, having an extremely fetid odor. When the softened material has been expectorated there is left behind a cavity with ragged walls, containing a foul-smelling liquid. The tissues around the cavity are inflamed and œdematous.

Symptoms.—The symptoms of gangrene are associated with the original disease. Cough, dyspnœa, moderate fever, and great prostration are generally present.

The expectoration is characteristic; it is profuse, and has a penetrating offensive odor. When allowed to stand in a glass

vessel it separates into three layers: a frothy layer on top, a serous layer in the middle, through which hang strings of pus, and at the bottom a layer of reddish-green purulent material. Altered blood may give it the appearance of prune-juice. Microscopically it contains shreds of tissue, crystals of fatty acids, crystals of hæmatoidin, and numerous pyogenic bacteria.

Physical examination may reveal bubbling râles, and later cavernous breathing, pectoriloquy, and localized tympany on percussion.

Prognosis.—Grave. Death usually results from exhaustion, but occasionally from hemorrhage or pyo-pneumothorax.

Treatment.—Nutritious food, and quinine, strychnia, and alcoholic stimulants will be required to support the system.

The offensive odor of the breath may be destroyed by carbolic acid (gr. j every four hours) internally, or by inhalations of carbolic acid or creosote. Turpentine (ɱv every three hours) has been recommended as a stimulant and antiseptic. When the patient's strength will permit, surgical interference offers the best chance of cure.

ABSCESS OF THE LUNG.

Definition.—Circumscribed suppuration of the lung.

Etiology.—(1) It is rarely a sequel to pneumonia. (2) Multiple abscesses are often embolic, and result from pyæmia. (3) Foreign bodies in the lungs—something swallowed or an hydatid cyst—may excite suppuration. (4) External abscesses sometimes rupture into the lung, as an empyema, hepatic abscess, or suppurating mastitis.

Symptoms.—High and irregular fever, rigors, sweats, and pallor indicate suppuration. Dyspnœa, cough, and purulent offensive sputa containing shreds of lung tissue are the pulmonary symptoms. Physical examination may reveal bubbling râles, and later, cavernous breathing and pectoriloquy. Multiple embolic abscesses are rarely recognized during life.

Prognosis.—Many cases following pneumonia and the rupture of external abscesses into the lung recover. Embolic abscesses generally prove fatal.

TREATMENT.—Nutritious food and quinine, strychnine, and alcoholic stimulants will be required to support the system. The abscess should be opened and drained, as the pleural sac is in empyema.

ŒDEMA OF THE LUNGS.

DEFINITION.—An effusion of serous fluid into the air-vesicles and into the interstitial tissue of the lungs.

ETIOLOGY.—Pulmonary œdema is a common cause of death in many acute and chronic diseases which end by heart-failure and the accumulation of blood in the lungs.

It is frequently noted in the course of Bright's disease and cardiac disease.

A local pulmonary œdema is often found around pulmonic consolidations, abscesses, and infarctions.

PATHOLOGY.—The lungs, especially the dependent portions, are heavy, red in color, and boggy to the feel. When the affected portion is incised and pressure is made, an abundant blood-stained, frothy serum exudes.

SYMPTOMS.—Extreme dyspnœa; rapid, labored breathing; cough with frothy, blood-stained expectoration; cyanosis; and cold extremities.

Physical Signs. *Inspection* reveals evidences of dyspnœa—sitting posture and prominence of the auxiliary muscles of respiration.

Percussion.—Dulness over the bases.

Auscultation.—Feeble respiratory murmur; subcrepitant or bubbling râles.

DIAGNOSIS. *Pneumonia.*—The absence of chill, of fever, of "rusty" tenacious sputa, of pain, and of signs indicating consolidation will indicate œdema.

Capillary Bronchitis.—The fever and muco-purulent expectoration will serve to distinguish bronchitis from œdema.

PROGNOSIS.—Always grave. It is often a final symptom of some pulmonary disease. When not advanced, and the conditions are favorable, recovery may follow.

TREATMENT.—When there is much cyanosis, and the patient's strength will permit it, the application of wet cups

to the chest or bleeding from the arm is of great value. Hot fomentations should be applied to the chest. Hydragogue cathartics are indicated. Epsom salts in concentrated solutions, or elaterium (gr. $\frac{1}{8}$), may be selected. Cardiac stimulants like ether, alcohol, ammonia, digitalis, and especially strychnine, are required, and may be given hypodermically.

℞ Strychnin. sulph., gr. j;
Aquæ destillat., f℥j.
Solve et sig.—15 minims hypodermically every three or four hours.

Caffeine is a useful diuretic, and cardiac and respiratory stimulant.

℞ Caffein. citratis, gr. xl;
Sodii benzoat., ʒiss.—M.
Ft. in chart. No. xii.
Sig.—One every two or three hours

PULMONARY COLLAPSE.

(Atelectasis.)

Definition—An absence of air from a portion of the lung.

Etiology.—It may be congenital and result from deficient respiration; in these cases the dependent portions of both lungs are commonly affected. Acquired atelectasis results from occlusion of a bronchus by a foreign body or a plug of mucus, as in capillary bronchitis; or from compression of the lung by a tumor or pleural effusion.

Symptoms.—When a large area is collapsed in some pre-existing disease like capillary bronchitis, there is an abrupt increase in the dyspnœa and cyanosis, without a corresponding rise of temperature. Physical examination gives negative results except over extensive collapse, which may give dulness on percussion and weak breathing on auscultation.

Prognosis.—This depends upon the extent of collapse and the gravity of the pre-existing disease.

Treatment.—In congenital atelectasis apply alternately hot and cold sponges to the spine; keep up the external temperature. If these measures fail, gently inflate the lung with a catheter.

In the acquired varieties direct remedies to the original

disease. Administer cardiac and respiratory stimulants like ammonia, strychnine, and nitroglycerine. When obstruction is due to a plug of mucus an emetic is indicated.

PULMONARY TUBERCULOSIS.

(Phthisis, Pulmonary Consumption.)

DEFINITION.—A specific inflammatory disease of the lungs, caused by the bacillus tuberculosis; characterized anatomically by a cellular infiltration which subsequently caseates, softens, and leads to ulceration of the lung tissue; and manifested clinically by wasting, exhaustion, fever, and cough.

ETIOLOGY.—(1) Residence in low, damp, and badly-drained localities. (2) Heredity (important). (3) Age; all ages, but especially between twenty and thirty years. (4) Occupations which necessitate the breathing of impure air and the inhalation of irritating dusts. (5) Catarrhal inflammation and traumatism of the lungs. (6) Physique. (7) General diseases which lower the vitality, as diabetes, hepatic cirrhosis, and typhoid fever.

The exciting cause is the bacillus tubercuIosis, which gains entrance (1) by direct parental transmission (very rare); (2) by inhalation, the dust of dried sputum being commonly the medium of contagion; (3) through infected food, as the milk and meat of tuberculous cattle.

VARIETIES.—(1) Chronic ulcerative phthisis. (2) Acute phthisis. (3) Fibroid phthisis.

PATHOLOGY.—The bacillus tuberculosis is a very minute rod, about one-fourth or one-half the diameter of a red blood-corpuscle, and often slightly bent and beaded. Its detection depends on the power of the stained bacillus to resist the decolorizing effects of acids. For satisfactory examination a one-twelfth oil-immersion lens is required.

The lodgment of bacilli in the terminal bronchioles of the apex excites a proliferation of the fixed cells, which become more or less polygonal in shape. The new cells are termed epithelioid, and frequently contain bacilli. Giant cells are often formed by a fusion or overgrowth of these cells.

This aggregation of new cells acts as an irritant and is soon

surrounded by a wall of leucocytes, the whole forming a gray, translucent mass—the gray tubercle of Laennec. In a short time the bacilli excite a coagulation-necrosis which starts in the centre, spreads to the periphery, and converts the tubercle into a yellow, cheesy mass—the yellow tubercle of Laennec. The degenerated tubercles fuse and form the uniform cheesy masses so commonly observed at the autopsy. At this stage one of two things may occur: The mass may soften, break into a bronchial tube, and leave behind a cavity with ulcerating walls, or it may become encapsulated by an overgrowth of connective tissue and subsequently calcified. In addition to the specific process other secondary changes are noted. The lung tissue in the neighborhood of the tuberculous deposits is often the seat of a true pneumonic inflammation; the connective tissue is always more or less proliferated; the bronchial tubes are inflamed; and the pleuræ over the affected areas are nearly always adherent.

Chronic ulcerative phthisis usually begins at the apices.

Acute phthisis has been termed *phthisis florida, cheesy pneumonia,* and *chronic catarrhal pneumonia,* but the process is invariably tuberculous. From extreme vulnerability of the tissues a lobe or whole lung, or even both lungs, are rapidly infiltrated, and death results in from a few weeks to a few months.

In some cases the lung is solidified by a dense yellowish-gray infiltration composed of closely-aggregated tubercles; in others the consolidation appears in more or less discrete patches which have had their origin in the smaller bronchial tubes; in a third form one or both lungs are studded with discrete tubercles, many of which are still gray and translucent.

In *fibroid phthisis* the tissues appear to be resistant, and the process is limited by an overgrowth of connective tissue which forms dense bands around the tuberculous foci. This form lasts many years.

Chronic Ulcerative Phthisis. Symptoms.—The onset is usually insidious and marked by pallor, gastric disturbance, loss of flesh and strength, and by a dry, hacking cough which is especially noted in the morning. From some undue exposure, the cough is often aggravated, and to this obstinate

"cold" the disease is usually attributed. In some cases, the symptoms appear abruptly with hemorrhage or an acute pleurisy.

Slight fever and acceleration of the pulse are early symptoms of great diagnostic import. The temperature is marked by an evening exacerbation, during which the face is flushed, the eyes bright, and the mind animated. As the disease advances the cough becomes troublesome and the expectoration more abundant. In well-developed cases the expectoration is greenish in color, is in coin-shaped plugs (nummular), is heavy and sinks in water, is often blood-streaked, and on microscopic examination is found to contain bacilli and fibres of elastic tissue.

Phthisis is in itself not a painful disease, but the associated dry pleurisy often causes much suffering. Hæmoptysis occurs at all stages, but the profuse hemorrhages occur late. The blood is bright red in color, frothy, and mixed with mucus. Dyspnœa is not a marked symptom, and its absence is doubtless due to the gradual development of the disease. Profuse sweating during sleep is a troublesome feature of advanced phthisis.

The final stage is characterized by extreme emaciation, weakness, pallor, high remittent or intermittent fever, and œdema of the feet. The mind is usually clear, and peculiarly hopeful to the end.

Physical Signs. *Inspection.*—The chest is usually long and flat; the spaces above and below the clavicles are sunken; the scapulæ are prominent; and the ribs are oblique.

There may be flattening or less expansion over one apex.

Palpation.—Diminished expansion and increased vocal fremitus.

Percussion.—Dulness, as a rule; this is noted earliest above or below the clavicles, in the supraspinous fossæ, between the scapulæ, or in front near the sternal border.

A cavity, or vomica, yields tympany, or a "cracked-pot" resonance. The latter can be more clearly demonstrated when the ear is placed near the patient's open mouth.

Auscultation.—In the early stage respiration may be inaudible over the affected area. Later the breathing is harsh

and the expiration prolonged and high-pitched (bronchial). The vocal resonance is increased. Crackling râles are usually audible, and are produced by liquid in the small tubes. If not present, coughing will usually develop them. Auscultation over cavities may detect cavernous or amphoric breathing, pectoriloquy, and large gurgling râles.

Anomalous Physical Signs.—The vocal fremitus is diminished when there is much pleural thickening. Normal resonance or hyper-resonance may replace dulness when there is much emphysema between small tuberculous foci. Weak breathing may replace bronchial or cavernous when the tubes or cavity are filled with muco-pus. The signs of cavity are sometimes produced by consolidation in the neighborhood of a large bronchus.

Acute Phthisis.—Clinically this form resembles pneumonia, and is marked by a chill, high fever, rapid pulse, dyspnœa, sputum at first rusty and then purulent, flushed face, profuse sweats, and the signs of consolidation. Instead of ending by crisis at the eighth or ninth day as an ordinary pneumonia, the symptoms grow rapidly worse, signs of softening appear, the sputum shows bacilli and elastic fibres, and death results in from a few weeks to a few months.

Fibroid Phthisis.—This is a disease of long duration. It is characterized by very gradual loss of flesh and strength and by an abundant muco-purulent expectoration, which is at times fetid from being retained in dilated bronchi. Dyspnœa, sweating, and fever are slight. There is very marked retraction on the affected side from the shrinking of the fibrous tissue; with this exception the physical signs are similar to those of ulcerative phthisis.

Complications of Phthisis.—Hæmoptysis; pneumonia; pleurisy; pneumothorax; stomatitis; obstinate vomiting induced by cough; diarrhœa; amyloid degeneration of the viscera; fistula in ano (tuberculous); and secondary tuberculosis of other organs, especially the larynx, cerebral meninges, and peritoneum.

Diagnosis.—Fever, cough, hæmoptysis, night-sweats, emaciation, signs of consolidation, and bacilli and elastic fibres in the sputum are the diagnostic phenomena.

Prognosis.—Generally unfavorable, though the disease is not incurable. The accidental discovery of calcified tubercles at autopsies furnishes abundant evidence of spontaneous cure. Many improve and a few recover under well-directed treatment.

A strong hereditary tendency, a bad physique, high fever, advanced consolidation, involvement of both lungs, even if slight, unfavorable surroundings, and, it might be added, a slender purse, render the prognosis extremely grave.

Treatment. *Preventive.* — Recognizing the infectious nature of the disease, the following prophylactic measures should be observed: Sputa of consumptives should be received in suitable vessels containing antiseptic solutions, and subsequently destroyed. Cattle should be rigidly inspected, and tuberculous meat, and milk of tuberculous cows declared unmarketable. Phthisical mothers should not nurse their offspring. The healthy should not sleep in apartments occupied by those affected.

Personal Hygiene.—Good food, fresh air, frequent bathing, avoidance of exposure, graduated exercise, residence in an elevated locality, a dry, well-ventilated house, and plenty of sleep and recreation.

Curative Treatment.—This involves two objects: (1) The strengthening of the patient's vitality and resisting power. (2) The destruction or disabling of the tubercle bacilli.

General Health.—The diet should be carefully regulated. Nutrients like cod-liver oil (℥ij—℥iv two hours after meals), malt, and hypophosphites are often very useful. Mineral acids and bitters may be required to stimulate digestion. Iron, quinine, and arsenic are sometimes indicated; the last, when well borne, often exerts a decidedly favorable influence. Alcohol in many cases is of great value, but the danger of inducing the habit must be borne in mind. Beer, porter, ale, and wine are usually the most desirable preparations. So long as alcohol stimulates the appetite, lowers the temperature, and strengthens the pulse it does good. Its results should be carefully noted, and any untoward effects will call for its immediate withdrawal.

Change of Climate.—This offers to many patients the greatest hope of cure. As a rule, a high altitude should be selected; the atmosphere should be dry and the temperature

equable. Personal experience must decide the question of temperature; generally, patients who feel better in summer will do well in a warm climate, and *vice versa*. The physician should have some knowledge of the locality, which should afford ordinary conveniences, without being too crowded with sufferers similarly afflicted.

In selected cases, a sea voyage is often very useful. According to Douglas Powell, it is most suitable to patients in the early stages, who have been previously healthy, who have overworked nervous systems, and in whom the disease is more or less quiescent.

Specific Treatment.—The injection of iodine, carbolic acid, etc. into phthisical lungs, as recommended by Mosler, Thompson, and Pepper, has not given encouraging results. Koch's tuberculin has been shown to be either negative or deleterious in its effects. Of the special remedies which have been recommended, creosote or one of its derivatives alone holds a prominent position in the therapy of phthisis. It may be given in pill, in emulsion of cod-liver oil, or with wine.

℞ Creosoti, ♏xv;
Olei morrhuæ, f℥iij;
Calcii et sodii hyposphos., ʒss;
Olei gaultheriæ, ♏xx;
Acaciæ, q. s.
Aquæ, q. s. ad f℥vj.—M.
Sig.—A tablespoonful two hours after meals.

The carbonate of guaiacol, being odorless and tasteless, and less irritating than creosote, is preferable to the latter. The daily dose is 15 to 60 grains.

℞ Strychnin. sulph., gr. ss;
Codein., gr. v;
Guaiacol carbonat., gr. c.—M.
Pone in capsulas No. xx.
Sig.—One every three hours.

Creosote is often valuable in inhalations.

℞ Creosoti,
Spt. chloroformi,
Alcoholis, āā f℥ss.—M.
Sig.—Ten to twenty drops in the inhaler several times daily.

Symptomatic Treatment. Cough.—Syrups should be avoided as far as possible, and cough alleviated by inhalations of wine of ipecac, creosote, benzoin, or terebene.

Tar, terebene, and eucalyptus may be employed internally. Cough associated with the expectoration of much offensive material should not be checked.

A cold bed often leads to cough and a wakeful night; in these cases the bed should be warmed before it is occupied. Hot applications to the chest and a hot drink on retiring sometimes insure rest.

The following mixture is very efficient in the cough of phthisis:—

℞ Codeinæ sulph., gr. iv;
Acid. hydrocyanic. dil., ♏xxxij;
Syr. tolu., f℥ij.—M. (Da Costa.)

Sig.—A teaspoonful three or four times daily.

Sweating.—Atropine (gr. $\frac{1}{100}$), picrotoxin (gr. $\frac{1}{80}$–$\frac{1}{50}$), gallic acid (gr. x), camphoric acid (gr. xx–xxx), agaricin (gr. $\frac{1}{4}$–1).

℞ Atropin. sulph., gr. $\frac{1}{4}$;
Acid. sulph. aromat., f℥ij;
Aquæ rosæ, q. s. ad f℥j.—M.

Sig.—Twenty to thirty drops at bedtime, and repeated if necessary.

Sponging with alum and whiskey is sometimes very efficacious.

Hæmoptysis.—When profuse, ice may be held in the mouth and swallowed slowly. The fluid extract of ergot (gtt. xx–xxx) and morphine (g. $\frac{1}{8}$) should be given hypodermically. The internal administration of gallic acid and other astringents is of little value. The application of a temporary ligature to one or more of the members hinders the flow of blood in the veins, and may materially aid in checking the bleeding.

When the hemorrhage is more or less continuous, but not profuse, the fluid extract of hamamelis (℥ij–℥iij) or pills of acetate of lead and opium are efficient remedies.

Diarrhœa.—Rest; liquid diet; subnitrate of bismuth in large doses, or pills of nitrate of silver and opium.

℞ Bismuth. subnit., ℥vj;
Salol, gr. xxiv;
Morphin. sulph., gr. j.—M.

Ft. in chart. No. xii.

Sig.—One powder every three hours.

Pyrexia.—Rest is imperative. Quinine or antipyrin, or sponging with alcohol and cool water, may prove useful. Guaiacol (10–20 drops) applied externally has been advocated, but its use is often followed by chills, sweating, and even collapse.

Pain.—The pleuritic pains may be relieved by opium and the application of adhesive strips, dry cups, or iodine.

PLEURISY.

(Pleuritis.)

Definition.—Inflammation of the pleura.

Varieties.—According to cause, it may be divided into primary or secondary; according to extent, into unilateral, bilateral, or local; according to time, into acute or chronic; and according to the exudation, into sero-fibrinous, fibrinous, or purulent.

Etiology.—Pleurisy may be: (1) Idiopathic, arising from exposure to cold and wet. (2) Traumatic. (3) Secondary to inflammatory diseases of adjacent viscera, as pneumonia and phthisis. (4) Secondary to some general morbid process, as rheumatism, Bright's disease, tuberculosis, and the infectious fevers. (5) Tuberculous. (6) Cancerous (rare).

Pathology.—In the early stage the membrane is red, sticky, lustreless, and covered with a thin film of lymph; if the process now ceases, the condition is termed *dry pleurisy.* If, however, the inflammation continues, an exudate is formed which may be: (1) Sero-fibrinous, (2) fibrinous, or (3) purulent (empyema). In the *sero-fibrinous* form there is little lymph, the exudate being mainly composed of straw-colored serum (a few ounces to several pints) which in favorable cases is gradually absorbed. In large effusions the adjacent organs are displaced and the lungs are compressed. In the *fibrinous* form serum is scant and the membrane is covered with a butter-like exudate which subsequently organizes and unites more or less closely the pleural surfaces, causing *adhesive pleurisy.* A liquid effusion, which is circumscribed and confined to pockets formed of adhesions, is termed *sacculated pleurisy.*

In the *purulent form* the sac is more or less filled with greenish-yellow pus. Purulent pleurisy, or *empyema*, is common in children; it frequently follows the infectious fevers; it is often secondary to a sero-fibrinous pleurisy; it results from the rupture of purulent accumulations into the pleura, as by a tuberculous cavity; and finally, it may be due to traumatism, as a penetrating wound or fracture of the ribs.

A purulent effusion left to itself may kill by sepsis, may become inspissated and encysted (rare), or may perforate into the bronchi, into neighboring organs, or externally.

Hemorrhagic Pleurisy.—A bloody effusion is observed in tuberculous and cancerous pleurisies and in pleurisy which is associated with scurvy, grave anæmia, and other cachectic states.

An effusion of any kind remaining unabsorbed constitutes a *chronic pleurisy.*

Symptoms. *Acute Pleurisy.*—The disease usually begins abruptly with a sharp, stabbing pain in the side and moderate fever (102°–103°). In some instances these symptoms are preceded by a chill. Cough appears early; it is usually dry, and, on account of the pain, it is partially suppressed. As the effusion accumulates and the inflamed surfaces separate, the pain diminishes, but dyspnœa and cyanosis rapidly develop.

Physical Signs. *First Stage.* — Less expansion on the affected side on account of the pain; occasionally a friction-fremitus on palpation, and a harsh to-and-fro friction-rub on auscultation.

Stage of Effusion. Inspection.—Immobility and bulging of the intercostal spaces on the affected side. The apex-beat is displaced upwards, and to the left or right according to the pleura affected.

Palpation.—Immobility and diminished vocal fremitus.

Percussion.—Dulness gradually rising as the fluid increases. The upper line of dulness is not horizontal, but is curved and rises higher posteriorly. In moderate effusions the level of dulness often changes with the position of the patient. Above the effusion percussion gives a tympanitic note which has been termed Skoda's resonance.

Auscultation.—The respiratory sounds are weak and distant; they may have a tubular or bronchial quality. The vocal resonance is usually diminished or absent, but occasionally bronchophony, or its modification ægophony (a bleating sound), is heard over moderate effusions.

Mensuration.—The affected side is sometimes an inch or more larger than the sound one.

After absorption of the effusion the friction-sound returns.

DIAGNOSIS. *Pneumonia.*—The severe chill, rusty expectoration, high fever, marked dyspnœa, the fine crepitant râles which are heard only on inspiration, dulness not changing with the patient's posture, increased vocal fremitus, increased vocal resonance, loud bronchial breathing, and the absence of bulging and of a displaced apex-beat, will serve to distinguish it from pleurisy.

Pleurodynia, or Rheumatism of the Intercostal Muscles.—No fever, much diffuse tenderness, no friction-sounds, and no effusion.

Purulent pleurisy is recognized by hectic symptoms—high and irregular fever, sweats, chills, and anæmia; by the results of aspiration; and sometimes by "pitting" from œdema of the surface.

Fibrinous Pleurisy.—Pain is severe and continuous, the dulness is immobile, aspiration gives negative results, and later there is much retraction of the affected side.

Tuberculous Pleurisy.—Tuberculosis is the most common cause of pleurisy which is apparently primary. It may be primary or secondary to pulmonary phthisis. It usually presents the same symptoms as ordinary sero-fibrinous pleurisy, but it often develops insidiously, is frequently bilateral, and the effusion is apt to be bloody. These facts, together with the history, will usually indicate the diagnosis.

Hydrothorax.—In this condition pain, fever, and friction-sounds are absent. The effusion is more apt to be bilateral. There is often a history of cardiac or renal disease, and the fluid on aspiration is found to contain less than 3 per cent. of albumin, and to have a specific gravity below 1.015.

Diaphragmatic pleurisy, or inflammation of the diaphragmatic pleura, may present the following symptoms: Intense

pain under the margin of the ribs, with tenderness on pressure; thoracic breathing; tenderness over the phrenic nerve, which is accessible between the two roots of the sterno-cleido-mastoid at the base of the neck; hiccough; and extreme dyspnœa. The physical signs are not marked.

PROGNOSIS.—This depends largely on the character and the amount of effusion. In primary sero-fibrinous pleurisy, the prognosis is usually good, but that pleurisies, which are apparently primary, are often tuberculous, should always be borne in mind. In purulent pleurisy, the prognosis is grave, though recovery frequently occurs.

In the fibrinous form, the prognosis is good, but if there has been much exudate, subsequent retraction and more or less impairment of the affected side are sure to follow.

TREATMENT.—Absolute rest. Light diet. If the temperature is high and the pulse rapid, aconite may be administered in small doses. Quinine (gr. v thrice daily) will exert a favorable influence. Pain may be so severe as to require morphia hypodermically.

Local Applications.—When the pain is severe, leeches or wet-cups, followed by strapping of the chest, will give great relief. In other cases, mustard plasters, hot fomentations, or iodine may be applied.

Serous Effusion.—Apply, frequently, small blisters. Iodide of potassium (gr. v thrice daily) may be employed for its absorbent effect.

Encourage diuresis with digitalis, caffeine, or acetate of potassium:—

℞ Potass. acetat., ℥ss;
Infus. digitalis, f℥iij.—M.
Sig.—Two teaspoonfuls every three or four hours.

Encourage catharsis with compound jalap powder (gr. xx–xxx) or Epsom salts.

℞ Magnesii sulphat., ℥iv–℥vj.
Div. in chart. No. viii.
Sig.—One powder in two tablespoonfuls of water before food, and no fluids for some time afterwards.

The effusion will require aspiration under the following

conditions: (1) When it excites much dyspnœa; (2) when it is very large, beyond the third or fourth rib; (3) when it is purulent; (4) when it remains unabsorbed after three or four weeks of careful treatment; (5) when it is bilateral, and the total amount is sufficient to fill one cavity.

The Operation.—Anæsthetize a point in the seventh interspace near the posterior axillary line and introduce the needle with a quick stroke along the upper border of the rib. The effusion should be drawn off slowly, and one or two pints removed according to the amount of the exudate.

Coughing during the operation is an indication for the withdrawal of the needle.

HYDROTHORAX.

Definition.—Thoracic dropsy.

Etiology.—It is always secondary, and may result from one of the causes of general dropsy, namely: Bright's disease, heart disease, emphysema or anæmia, or from the pressure of a tumor or aneurism upon the thoracic veins.

Symptoms.—Dyspnœa, cyanosis, and the physical signs of a pleural effusion.

Diagnosis. *Pulmonary Œdema.*—The upper level of dulness is not movable; the intercostal spaces are not unduly prominent; the apex-beat is not displaced; and auscultation reveals abundant moist râles.

Treatment.—Remedies should be directed to the original disease. When there is much dyspnœa, aspirate.

PNEUMOTHORAX.

Definition.—Air in the pleural sac.

Etiology.—It may result from: (1) The rupture of the lung in health from a violent strain, or rupture in tuberculosis, abscess, emphysema, or gangrene. (2) Traumatism, as a penetrating wound or a fracture of the ribs. (3) The rupture of an empyema into the lung.

Pathology.—The adjacent viscera are displaced, and the

lung is compressed. Even when air alone has escaped into the pleural sac an effusion soon develops, so that in all cases the condition becomes a *pneumo-hydrothorax* or *-pyothorax.*

Symptoms.—The onset is marked by a sharp pain, extreme dyspnœa, cyanosis, and symptoms of incipient collapse, namely, a fall of temperature, a weak rapid pulse, cold extremities, and pinched features.

Physical Signs. *Inspection.*—Immobility, and bulging of the intercostal spaces. The apex-beat is usually displaced.

Palpation.—Diminished vocal fremitus.

Percussion.—A tympanitic note, varying in pitch with the intrathoracic tension.

Effusion sinks to the base and yields dulness, the outline of which changes with the position of the patient.

Auscultation.—The respiratory murmur and vocal resonance are usually absent, but when the opening in the lung remains patulous, amphoric breathing may be detected. When a silver coin is placed on the affected side and is struck with another, the auscultator detects a clear metallic sound (bell-tympany). When fluid is present, shaking the patient excites a splashing sound (Hippocratic succussion).

Diagnosis. *A large Phthisical Cavity.*—This is usually located near the apex instead of the base; the surface is sunken, not prominent; the heart is not displaced; succussion-splash and bell-tympany are usually absent.

Dilated Stomach.—This may give a tympanitic note over the left pulmonary base, and may simulate a pneumothorax; but the tympanitic note is continued down into the abdomen, and the swallowing of liquid is distinctly audible over the base of the chest.

Prognosis.—It is usually unfavorable, and often terminates fatally in a few hours or days. Recovery is possible, especially in traumatic cases. It often excites a pleural effusion and runs a chronic course.

Treatment.—At the onset administer stimulants, and apply straps to the chest. The pain and distress must be relieved by morphine. When effusion forms it should be treated, according to its character, as a serous or a purulent pleurisy.

HÆMOTHORAX.

(Hæmatothorax.)

DEFINITION.—The effusion of blood into the pleural sac.

ETIOLOGY.—Traumatism, rupture of an aneurism, or the erosion of bloodvessels by cavities or caries of the ribs.

SYMPTOMS.—Same as hydrothorax.

TREATMENT.—When there is great dyspnœa the blood should be removed by aspiration or incision.

PYOTHORAX.

(Empyema.)

DEFINITION.—An effusion of pus into the pleural sac.

ETIOLOGY.—(1) The effusion may be primarily purulent, the inflammation having been excited by pyogenic microörganisms. (2) A sero-fibrinous pleurisy, through subsequent infection, may be converted into an empyema. The predisposing causes are much the same as those of sero-fibrinous pleurisy. Traumatism or the rupture of a purulent accumulation into the pleural sac is an occasional cause. It frequently follows pneumonia, particularly in children, in whom the most common form of pleurisy is empyema. It is often secondary to tuberculosis or one of the infectious fevers.

Streptococci, pneumococci, tubercle bacilli, Eberth's bacilli, and staphylococci are capable of exciting empyema.

SYMPTOMS.—The physical signs and symptoms are similar to those observed in sero-fibrinous pleurisy. Pus is indicated by hectic phenomena—high and irregular fever, sweats, chills, and anæmia; by the results of aspiration; and sometimes by œdema of the chest-walls. In pulsating pleurisy the effusion is almost always purulent.

PROGNOSIS.—Grave, though recovery frequently occurs. The most favorable cases are those following pneumonia.

TREATMENT.—Free incision and thorough drainage. Irrigation is unnecessary unless the fluid is putrid. In long-standing cases the excision of several ribs (Estlander's operation) facilitates retraction and the obliteration of the pleural sac, which is essential to a cure.

ACUTE INFECTIOUS DISEASES.

FEVER.

Fever is an abnormal condition characterized by elevated temperature, quickened respiration and circulation, faulty secretions, and increased tissue-waste; and dependent upon a perversion of the physiological processes whereby the generation and loss of heat are so balanced as to maintain a uniform normal temperature.

The Detection of Fever.—There is only one sure way of detecting fever, and that is by means of the clinical thermometer. The instrument may be placed in the axilla, mouth, rectum, or vagina.

When the axilla is selected the following precautions must be observed: Wipe off the perspiration and dry the skin; insert the bulb of the instrument deep in the armpit, and see that the arm is kept close to the side. The thermometer should be kept in position until the mercury maintains the same level for two minutes; this will usually require in all about six or seven minutes.

When the mouth is selected the bulb should be placed under the tongue and the lips kept closed. Hot and cold drinks recently taken mar the results. For obvious reasons the mouth should not be used in delirious patients.

The rectum may be selected in children. The rectal temperature is about a degree higher than that of the axilla.

Febrile Stages.—The course of all fevers is marked by three stages: (1) Invasion; (2) fastigium, or stadium; (3) defervescence, or decline.

Invasion.—During this period the temperature gradually rises until it reaches its maximum.

Fastigium.—In this period, though there may be marked variations, the temperature shows a tendency to touch again and again its highest point.

Defervescence.—In this period the temperature gradually falls until it reaches the norm.

Terminations of Fever. — Fever terminates by lysis or crisis.

Lysis.—The temperature falls slowly by slight gradations until it reaches the norm.

Crisis.—The temperature falls suddenly, often four or five degrees in twelve or twenty-four hours.

The Degree of Pyrexia.—The following is Wunderlich's classification of febrile temperatures :—

1. Subfebrile, temperature 99.5°–100.4°.
2. Slightly febrile, temperature 100.4°–101.3°.
3. Moderately febrile, temperature 101.3°–103.1°.
4. Decidedly febrile, temperature 103.1°–104°.
5. Highly febrile, temperature above 103.1° in the morning and above 104.9° in the evening.
6. Hyperpyretic, temperature above 106°.

Febrile Remissions.—All fevers show a diurnal variation. The maximum is usually reached at about 6 P.M. and the minimum at about 6 A.M. Sometimes these extremes are reversed and the maximum is in the morning and the minimum in the evening. The daily difference amounts to about 1°.

Types of Fever.—According to the degree of the diurnal variation three types are recognized :—

1. *Continued Fever.*—The diurnal variation is slight, 1°–1.5°. Typhus fever, pneumonia, and scarlet fever are examples of continued fevers.

2. *Remittent Fever.*—The diurnal variation is marked, but the minimum temperature is still above the norm. Typhoid fever, remittent fever, and hectic fever are examples of this type.

3. *Intermittent Fever.*—The diurnal variation is marked, and the minimum is normal or subnormal. The following fevers intermit :—

1. Intermittent fever (malaria).
2. Relapsing fever.
3. Hectic fever (often intermits, though generally remits).
4. Charcot's intermittent (the peculiar fever associated with the impaction of gall-stones).

Causes of Fever.—(1) Local inflammations excited by external causes, or the products of faulty metabolism (gout, rheumatism). (2) The presence in the body of micro-örganisms, or of toxines produced by them, as in typhoid fever, pyæmia, scarlet fever, etc. (3) Paralysis of the heat-centre, as in thermic fever.

Symptoms of Fever.—Rise of temperature; rapid pulse; rapid respirations; coated tongue; anorexia; constipation. The urine is scanty, high-colored, throws down a heavy sediment, and may contain a trace of albumin. The gastric juice is deficient in acid. If the fever is long-continued, the body wastes.

The Pulse-temperature ratio:—

A temperature of	98.4°	corresponds to a pulse of			70
" "	100°	"	"	"	80– 90
" "	102°	"	"	"	100–110
" "	104°	"	"	"	120–130

Effects of Fever on the Tissues.—High and long-continued fever produces fatty and parenchymatous degeneration of the tissues.

Treatment of Fever.—Absolute rest; a cool, well-ventilated room; liquid or semi-liquid diet. Slight fever will require no special remedies, but the patient may be made more comfortable by sponging with cool water, or water and alcohol; and by the use of such drugs as sweet spirits of nitre, acetate of ammonium, or neutral mixture.

High fever is best controlled by the external application of cold; this method includes sponging with cold water, the cold pack, and the cold bath.

The Cold Pack.—A rubber sheet is slipped under the patient, and the body is enveloped in a sheet wrung out in cold water,

which is allowed to remain until the temperature falls one or two degrees.

The Cold Bath.—There are two methods of administering the cold bath. The first is to place the patient at once into water at 70°; the other is to place him into water at 90° or 80°, and then gradually cool it down to 75° or 70°. While in the water an ice-bag is kept upon his head, and his body is subjected to vigorous rubbing. He should remain in the bath for fifteen or twenty minutes, after which he should be placed in a dry sheet and covered with a light blanket. When the body is dry the damp sheet should be removed. A stimulant is sometimes required during or after the bath.

Drugs may be employed to lower temperature, but the bath is preferable when it is feasible. Quinine, antipyrin, phenacetin, and acetanilid are the antipyretics most commonly employed.

Period of Incubation.—The period elapsing between the entrance of the poison and the development of symptoms.

It varies considerably in the same disease, being more or less influenced by the susceptibility of the patient and the virulence of the contagion. The average period of incubation in the infectious fevers is as follows:—

Typhoid fever: two to three weeks.
Typhus fever: a few hours to two weeks.
Measles: two weeks.
Rötheln or rubella: ten to twelve days.
Scarlatina: a few hours to a week.
Smallpox: one to two weeks.
Erysipelas: three to seven days.
Diphtheria: two to ten days.
Varicella: ten to fifteen days.
Tetanus: a few days to two weeks.
Mumps: two to three weeks.
Yellow fever: from a few hours to a week.

The date at which rashes appear in the various diseases:—

Typhoid fever: seventh to the ninth day.
Typhus fever: fourth or fifth day.

Smallpox: third or fourth day.
Measles: third or fourth day.
Scarlatina: first or second day.
Rötheln or rubella: first or second day.
Varicella: first day.

Protection from Future Attacks.—Few diseases give absolute immunity from future attacks, but the following are fairly protective:—

Typhoid fever: relapses are common, and second attacks sometimes occur.
Typhus fever: second attacks very rare.
Measles: second attacks uncommon; what is supposed to be a second attack is usually rötheln.
Rötheln: second attacks uncommon.
Scarlatina: second attacks rare.
Smallpox: second attacks occasionally occur.
Mumps: second attacks rare.
Yellow fever: second attacks rare.

The following do not confer immunity:—

Erysipelas.	Malaria.
Relapsing fever.	Influenza.
Diphtheria.	Croupous pneumonia.

Periodic Remission or Intermissions in the Fever.—Such remissions or intermissions occur in the following fevers:—

Malarial fever: every day, every third day, or every fourth day, according to the type.
Relapsing fever: intermissions occur at intervals of five or six days, and last five or six days.
Smallpox: remission occurs on the third day.
Measles: a distinct remission often occurs on the second or third day.
Yellow fever: a marked remission on the third or fourth day.
Dengue: a marked remission on the third or fourth day, which lasts two or three days.

The Infectious Fevers which are usually Associated with Jaundice:—

Yellow fever.
Relapsing fever.
Acute yellow atrophy of the liver.
Bilious remittent fever.

Termination by Crisis.—The following infectious fevers are apt to end by crisis:—

Typhus fever.
Pneumonia.
Influenza.
Measles.
Relapsing fever.
Erysipelas.

SUBNORMAL TEMPERATURE.

Temperatures below 97.5° may be considered subnormal. They are observed in the following conditions:—

1. During convalescence from certain febrile diseases; after pneumonia and typhoid fever the temperature may remain subnormal for several days.

2. In collapse. This may result from shock; from hemorrhage; from the action of some toxic agent; from simple heart-failure in the course of disease; or from the rupture of a viscus, as the bowel in typhoid, the lung in phthisis, or the stomach in perforating ulcer.

3. In cholera. In this disease the temperature may be very low (90°–85°) for several days.

4. In certain chronic diseases, especially myxœdema, diabetes, cancer, chronic cardiac, cerebral, and spinal diseases.

SIMPLE CONTINUED FEVER.

(Febricula, Ephemeral Fever.)

Definition.—An acute febrile disease, of short duration, and not excited by a special poison.

Etiology.—It is generally met with in young and sensitive individuals. Exposure to the sun, prolonged physical or emotional excitement, and errors in diet seem to excite it.

Symptoms.—The disease usually begins abruptly with chilliness, headache, malaise, and fever which soon attains a maximum of 102° or 103°. The face is flushed; the pulse is full and rapid; the urine is scanty and high colored; the tongue is coated; the appetite is lost; and the bowels are constipated. There is no characteristic eruption, but herpes is frequently observed on the lips.

The disease lasts from a few days to two weeks, and may end by crisis or lysis.

Diagnosis.—Care must be taken to exclude local inflammations, such as gastritis, tonsillitis, and pneumonia.

Typhoid Fever.—At first the diagnosis may be impossible, but the absence of diarrhœa, tympanites, abdominal tenderness, splenic enlargement, and eruption will soon make the diagnosis apparent.

Remittent Fever.—The history, locality, splenic enlargement, and hæmatozoa in the blood will serve to distinguish this disease from simple continued fever.

Prognosis.—Favorable.

Treatment.—Absolute rest in bed. A liquid diet. Repeated small doses of calomel may be employed to relieve the constipation.

The fever may be controlled by sponging with water and alcohol or by the use of some mild refrigerant mixture like the following:—

> Tinct. aconit. rad., gtt. iij;
> Spt. æther. nitrosi, f℥ss;
> Liquor. ammon. acetat., q. s. ad f℥iij.—M.

Sig.—A dessertspoonful every two hours to a child of four years.

TYPHOID FEVER.

(Enteric Fever, Typhus Abdominalis.)

DEFINITION.—An acute infectious disease, excited by a special bacillus, characterized anatomically by definite lesions in Peyer's patches, mesenteric glands, and spleen; and manifested clinically by fever, headache, stupor, abdominal distention and tenderness, diarrhœa, enlargement of the spleen, and a rose-colored abdominal rash.

ETIOLOGY.—Predisposing causes: Autumn season, early adult life, and a personal susceptibility.

Exciting cause: The bacillus of Eberth. The intestinal discharges are the source of the contagion, and drinking-water contaminated by them becomes the chief medium of transmission.

PATHOLOGY.—The characteristic lesions are found in the abdominal lymphatics, namely, in Peyer's patches, solitary glands, and mesenteric glands. The changes in Peyer's glands are best studied in the lower part of the ileum, which should be opened on the side of the mesenteric attachment.

In the first few days the glands are swollen and hyperæmic; later there is a marked cell-proliferation, the bloodvessels are compressed, and the glands become pale and prominent (medullary infiltration). If the disease advances, necrosis sets in about the second week; the glands become yellow and soft and discharge their contents, leaving behind irregular oval ulcers with swollen and undermined edges, and with smooth bases formed by the submucous coat, muscular coat, or peritoneum. In the fourth week cicatrization begins, and the gland is ultimately replaced by a smooth depressed scar.

In addition to these glandular lesions, the mucous membrane of both large and small intestines shows catarrhal changes.

In mild cases the stage of ulceration may not be reached, the proliferated cells being removed by fatty degeneration and absorption without rupture of the gland. The solitary and mesenteric glands pass through similar changes, but the latter rarely rupture. Other lesions are found which are not characteristic. The spleen is soft and swollen, and occasionally rup-

tures. The liver, kidneys, heart, and muscles reveal parenchymatous degeneration. The respiratory tract is commonly the seat of catarrhal inflammation. In rare instances there appears to be a general infection without lesions of the intestinal glands (typhoid septicæmia).

Period of Incubation.—Two to three weeks.

Fig. 17.

Temperature curve in typhoid fever.

Symptoms. *Prodromal Symptoms.*—Gradual weakness, headache, vague pains, nose-bleed, and often slight diarrhœa.

The Attack. Fever.—The temperature rises gradually, reaching a maximum (104°–105°) in from one to two weeks; it remains at this elevation for another period of from one to two weeks, when a gradual defervescence begins and occupies a third period lasting from one to two weeks. Throughout its course the fever is characterized by marked daily remissions, the evening temperature being from one to three degrees higher than the morning.

In some cases, especially in the young, the temperature rises quite abruptly. Slight diurnal remissions indicate a protracted case. As defervescence advances, the temperature becomes more irregular; the remissions are more decided, and not infrequently the higher temperature is recorded in the morning. An abrupt fall of several degrees should suggest intestinal hemorrhage or perforation.

Respiratory Symptoms.—Hurried respirations, slight cough, and bronchial râles.

Circulatory System.—The pulse becomes rapid, weak, and dicrotic. The rapidity is often less than such temperatures

generally produce. The heart-sounds become feeble. The first is especially weak and resembles the second.

The Face.—The expression is dull and heavy, the cheeks are somewhat flushed, the conjunctivæ are clear, and the pupils dilated.

The tongue is tremulous; at first it is red at the tip and edges, and covered posteriorly with a whitish fur. In severe cases the tongue becomes dry, brown, and fissured, and *sordes* collect on the teeth.

The Stomach.—Gastric symptoms are not common, but obstinate vomiting sometimes develops and becomes a serious complication.

Intestinal Symptoms.—The belly is distended with gas. Tenderness is frequently noted on palpation; it may be general, or confined to the right iliac fossa. Gurgling may also be detected in the latter region, but it has little significance. Diarrhœa is generally present, though it is not a constant symptom. The discharges vary in number from three to six or more a day; they are thin, offensive, and of a yellowish color (likened to pea-soup); on standing, a turbid liquid rises to the top and a granular sediment falls to the bottom.

The Eruption.—This appears from the seventh to the ninth day, and is most abundant on the abdomen, though it is not infrequently observed on the chest and back. It is composed of small, slightly elevated, rose-colored spots which disappear on pressure. It comes out in successive crops over several days. It may be absent particularly in the old and very young. Rarely, in malignant cases, is the eruption petechial.

Sudamina are also noted, and result from free perspiration.

Splenic enlargement is rarely absent. The organ may rupture.

Nervous Symptoms.—Headache, slight deafness, stupor, muttering delirium, twitching of the tendons (subsultus tendinum), picking at the bedclothes or imaginary objects (carphologia), and coma vigil (the eyes are open, but the patient is unconscious).

The Blood.—An examination of the blood reveals a reduction in the number of both red and white cells.

Widal Reaction.—Blood-serum of typhoid patients when mixed with a fresh bouillon-culture of the typhoid bacillus,

after the lapse of a few hours, clears the liquid and throws down a flocculent precipitate. Microscopic examination shows that this precipitation is due to a loss of the motility of the bacilli and their agglutination or aggregation in clumps. The reaction does not appear, as a rule, before the end of the seventh or eighth day, and may persist for several months or years after recovery. It can be obtained from dried blood or from blood collected in a glass tube of small calibre. As a means of diagnosis it is reliable only when the serum is mixed with the bouillon-culture in no greater proportion than 1 to 40. Of 2283 typhoid cases, 95.57 per cent. yielded the reaction; of 1365 non-typhoid cases, there was no reaction in 98.4 per cent.

The urine is febrile and often slightly albuminous. Retention is common.

Convalescence is marked by anæmia, falling of the hair, desquamation of the cuticle, and often mental enfeeblement.

VARIETIES. *Mild Typhoid.*—There is moderate fever with marked remissions; the diarrhœa is slight; nervous symptoms are often absent; the rash is usually present, and often abundant.

Abortive Typhoid.—There is an abrupt onset with severe symptoms, but convalescence follows in a few days.

Walking Typhoid.—The symptoms are mild, and often disregarded by the patient, who refuses to go to bed; but grave symptoms may develop suddenly, and death from perforation is not uncommon.

Typhoid in Children.—The rash is often absent; the fever rises abruptly; cerebral symptoms are marked.

COMPLICATIONS.—Any symptom aggravated constitutes a complication; thus high fever, excessive diarrhœa, and tympanites become complications.

Hemorrhage.—This usually occurs during the third week, and is indicated by a sudden fall of temperature, followed by dark red or tarry stools.

Peritonitis.—This may result from perforation, or from extension by contiguity. The former is the more common, and is recognized by a sudden pain, a fall of temperture, distention of the belly, and symptoms of peritonitis.

Pneumonia and *hypostatic congestion of the lungs* are common complications.

Among less frequent complications or sequelæ may be mentioned: Nephritis, neuritis, suppurative cholecystitis, appendicitis, pyelitis, tuberculosis, temporary insanity, parotitis, and phlegmasia dolens.

Relapse and Recrudescence.—*Relapses* are quite common; they repeat the symptoms of the original attack, but they are generally milder and of shorter duration, and seldom prove fatal.

Recrudescence.—This is a sudden temporary elevation of temperature occurring during convalescence, and is not associated with a return of the other symptoms. It is usually due to constipation, excitement, or irritating food.

Diagnosis.—*Acute miliary tuberculosis* often closely resembles typhoid fever. In tuberculosis the temperature is generally more irregular; the abdominal symptoms are less marked; pulmonary symptoms, especially dyspnœa, are more marked; the rash is absent; tubercles may be detected on the retina; and symptoms of basilar meningitis may be present, such as irregular pupils, ptosis, and strabismus.

Ulcerative Endocarditis.—The diagnosis may be impossible, but the following features would suggest endocarditis: The history of a primary disease which might induce ulcerative endocarditis; irregular fever; intercurrent rigors; marked leucocytosis; præcordial pain and endocardial murmurs; and the absence of a rose-colored rash, of the Widal reaction, and of marked abdominal symptoms.

Enteritis.—The absence of high fever, of eruption, of splenic enlargement, of epistaxis, of bronchial catarrh will serve to distinguish enteritis from typhoid fever.

Meningitis.—The abrupt onset, the early development of cerebral symptoms, the irregular fever, and the absence of a rash and of abdominal symptoms will indicate meningitis.

Prognosis.—The prognosis should always be guarded. No case is too mild to prove fatal, and no case is too severe to recover. The mortality varies in different epidemics. In private practice the average is probably between five and ten per cent., and in hospital practice it is somewhat more.

Continued high fever with slight diurnal remissions, excessive diarrhœa, severe cerebral symptoms, and repeated hemorrhages are unfavorable features.

Treatment.—Absolute rest in bed and the enforced use of the bed-pan. The stools should be rendered innocuous. This may be done by dissolving a pound of chloride of lime in four gallons of water, and adding a quart of the solution to each discharge, and allowing it to remain in the vessel at least an hour before disposing of it. Soiled bedclothes should be thoroughly boiled.

The diet must be liquid, and preferably milk. From two to four pints should be given in the twenty-four hours, and should be so divided that the patient shall receive a small amount every two hours, day and night. When it causes eructations or flatulence, or is discharged undigested, it must be mixed with lime-water, or be predigested. Koumiss is often acceptable. Meat-broths may be given to vary the monotony of a milk diet. Cool water or ice will be required to allay thirst, and even if the latter is absent, it is well to give one or the other at regular intervals. When the first sound of the heart weakens and the pulse becomes soft, stimulants should be administered. It is desirable to give the alcohol with the milk so as to stimulate the stomach to digest the latter, and at the same time to diminish the number of administrations of food and medicine. From four to eight ounces of brandy or whiskey may be required in the twenty-four hours, the amount being determined by the general effect. When additional stimulation is required strychnine is a valuable adjunct.

When the tongue becomes dry and brown, the belly much distended, and low nervous symptoms develop, turpentine will be found an invaluable stimulant. Five to ten minims may be given in capsule or emulsion every two or four hours.

Antiseptic remedies have been strongly advocated, but their efficiency has not been clearly demonstrated. Thymol, naphthol, carbolic acid, chlorine-water, iodine, and calomel are the antiseptics which have been recommended.

The use of the cold bath or the cold pack will be found an excellent method of controlling fever and of preventing the

development of severe nervous symptoms. It is especially valuable as a stimulant to the nerve-centres, and may be employed whenever the temperature exceeds 102⅖°. Hemorrhage and perforation contraindicate its use. (See page 247.)

Fever.—When circumstances prevent the use of the cold bath, sponging with cool water and the administration of such antipyretics as quinine (gr. xx–xxx) or antipyrin (gr. v–x) may be substituted.

Diarrhœa.—When diarrhœa exceeds more than three or four stools a day, it is well to check it by an opium suppository, or by bismuth or nitrate of silver by the mouth.

℞ Pulv. opii, gr. iij ;
Ol. theobrom., q. s.—M.
Ft. in suppos. No. vi.
Sig.—One, two or three times daily.

Or—

℞ Morph. sulph., gr. j ;
Creosot., gtt. vj ;
Bismuth. subnit., ʒiij.—M.
Ft. in chart. No. xii.
Sig.—One every two or three hours.

Or—

℞ Argenti nit., gr. v ;
Ext. opii, gr. iv.—M.
Ft. in pil. No. xx.
Sig.—One every three hours.

Constipation.—This may be relieved by an enema of soap and water, or by broken doses of calomel.

Tympanites.—Turpentine stupes. Turpentine or thymol internally. In grave cases, rectal intubation.

Hemorrhage.—An ice-bag to the right iliac fossa. Morphine (gr. ⅛) with ergotine (gr. v–x) hypodermically. Turpentine or gallic acid may be administered by the mouth.

Perforative Peritonitis.—About 1 or 2 per cent. of typhoid cases end in perforation. This complication is almost invariably fatal. Morphine should be given freely. Operative interference offers some hope in selected cases. In 30 operations there were 6 recoveries.

Heart-failure.—When alcohol is being used in large amounts and the symptoms of heart-weakness still persist,

such remedies as aromatic spirits of ammonia, ether, strychnine, digitalis, or cocaine may prove useful.

Grave Nervous Symptoms.—Delirium, subsultus, insomnia, etc., are best controlled by cold bathing. Nerve sedatives, like the bromide of potassium, musk, hyoscine, sulphonal, and camphor, are sometimes required.

TYPHUS FEVER.

(Ship Fever, Jail Fever.)

Definition.—An acute contagious disease unassociated with any characteristic lesions of the solids, and manifested by great prostration, a petechial rash, marked nervous symptoms, and high fever which defervesces by crisis in from ten to fourteen days.

Etiology.—It is excited by an unknown poison which is capable of being carried in clothes (fomites). It is rare in America, but not uncommon in England and Ireland. Bad food, impure water, overcrowding, and foul air are predisposing factors.

Pathology.—There are no characteristic lesions of the solids. As in other fevers, the liver and spleen are swollen, and the tissues reveal fatty and parenchymatous degeneration. The blood shows a peculiar change: it is dark, fluid, and stains the lining of the heart and great bloodvessels bright red.

Period of Incubation.—A few hours to two weeks.

Symptoms.—Typhus fever begins abruptly with pain in the head, back, and limbs; extreme prostration; and fever which reaches its maximum (104°–105°) in two or three days. The temperature remains high for about ten days, when it falls by crisis.

The pulse is rapid, weak, and often dicrotic. The tongue is tremulous, and usually covered with a whitish fur; but in bad cases it becomes black and rolled up like a ball in the back of the mouth.

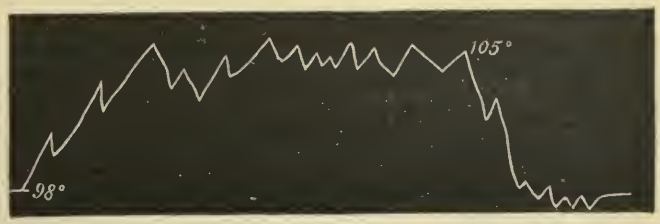
The face is dusky; the conjunctivæ are injected; and the pupils are contracted.

Nervous Symptoms.—These are prominent, and consist of

headache, stupor, delirium, subsultus tendinum, carphologia, and coma vigil.

The Eruption.—About the fourth or fifth day rose-colored spots appear over the body; these rapidly become hemorrhagic, or petechial, and fail to disappear on pressure. There is a

Fig. 18.



Temperature chart of typhus.

distinct relation between the amount of eruption and the severity of the attack. In addition to this "mulberry rash," there is often a diffuse, dark-red subcuticular mottling.

Gastro-intestinal Symptoms.—The stomach is retentive, and the bowels are constipated.

Urine.—The urine is scanty, high-colored, and often albuminous.

Complications.—Hyperpyrexia, catarrhal pneumonia, hypostatic congestion of the lungs, nephritis, and parotid abscess.

Diagnosis. *Cerebro-spinal Meningitis.*—In this affection the pain in the back is greater. The fever is very irregular; there is greater tendency to opisthotonos and facial palsies; and the eruption, though it may resemble that of typhus, is inconstant and without a special time for appearing.

Typhoid Fever.—The resemblance is in the nervous phenomena only. In typhoid the fever rises and falls very gradually; the eruption appears later, remains rose-red, and does not become petechial; the face is not dusky, the eyes are not injected; and there are marked abdominal symptoms.

Prognosis.—The mortality is much greater than in typhoid

fever. Advanced years and alcoholism render the prognosis decidedly unfavorable.

Treatment.—Isolation; absolute rest; liquid diet. There is no specific treatment. Alcohol is nearly always required. Quinine and mineral acids are useful tonics.

Pyrexia, *nervous phenomena*, and *heart-failure* should be treated as in typhoid fever.

RELAPSING FEVER.

(Spirillum Fever, Famine Fever.)

Definition.—An acute contagious disease excited by the spirochæte of Obermäier, and characterized by paroxysms of high fever which last five or six days and are followed by intermissions of a similar duration.

Etiology.—The exciting cause is the spirochæte of Obermäier, a spiral-shaped microbe three or four times as long as the diameter of a red blood-corpuscle. Bad water, poor food, overcrowding, and foul air predispose to epidemics. The disease is highly contagious.

Pathology.—There are no characteristic lesions. The liver and spleen are much enlarged, and the latter is frequently the seat of infarctions. There is usually catarrhal inflammation of the stomach and bile-ducts. The spirochæte is found in the blood during life, but only during the paroxysms; after death it is found in all the organs.

Period of Incubation.—Five to eight days.

Symptoms.—The disease begins abruptly with a chill followed by fever, which reaches its maximum (105°–106°) in twenty-four hours, and remains high for from five to seven days, when it falls by crisis. After an intermission of five or six days it again rises rapidly and remains high for a similar period. Convalescence usually *begins* at the end of the second paroxysm, but it may *not begin* until after the third or fourth. Other noteworthy symptoms are intense pains in the head, back, and limbs; the spirochæte in the blood; and frequently jaundice.

Complications.—Hyperpyrexia, nephritis, pneumonia, and ophthalmia.

Diagnosis. *Rheumatic Fever.*—The history, irregular fever, acid sweats, and the absence of spirilli and of jaundice will serve to distinguish rheumatism from relapsing fever.

Remittent Fever.—In this disease the fever remits, but does

Fig. 19.

Temperature curve in relapsing fever.

not intermit; the paroxysms are more frequent; and instead of spirilli, hæmatozoa are found in the blood.

Yellow Fever.—The single remission on the second or third day, the bloody vomit, and the absence of spirilli and of splenic enlargement will indicate yellow fever.

Prognosis.—Favorable in uncomplicated cases.

Treatment.—Isolation; rest; liquid diet. As a general tonic, quinine is useful. For the pains, antipyrin, phenacetin, or morphine may be given internally, and rubefacients used locally. For the irritable stomach hot fomentations may be applied to the epigastrium, and small doses of calomel and soda administered internally.

CEREBRO-SPINAL FEVER.

(Epidemic Cerebro-Spinal Meningitis, Spotted Fever.)

Definition.—A specific infectious disease characterized anatomically by inflammation of the cerebro-spinal meninges, and clinically by intense pain in the head, back, and limbs,

convulsions, irregular fever, and frequently by a petechial eruption.

Etiology.—The disease may be sporadic or epidemic. Overcrowding, poor food, foul air, and bad drinking-water seem to predispose to epidemics. Outbreaks are most common in the winter and spring. The young are more susceptible than the old. The disease is not contagious; the method of transmission is still unknown.

The Exciting Cause.—This is the diplococcus intracellularis of Weichselbaum. In the tissues this organism is found chiefly in the leucocytes of the exudation.

Pathology.—In most cases the membranes of the brain and cord are deeply congested and opaque. Lymph and pus are found both at the base and on the convexity of the brain, especially in the fissures and along the bloodvessels. The spinal meninges present similar changes, the posterior surface of the cord being particularly involved.

The liver and spleen are engorged and the muscles reveal granular degeneration. In rapidly fatal cases the lesions are very slight.

Symptoms. **Common Form.**—The disease generally begins abruptly with a chill, followed by vomiting and excruciating pain in the head, back, and limbs. The muscles of the neck and back become rigid and contracted, so that the head is bent backward and the back is straightened; in severe cases the body may be arched in a state of opisthotonos. The mind is soon affected; delirium is rarely absent, and in severe cases it is followed by stupor and coma.

Involvement of the Cranial Nerves.—Pressure of the exudate upon the cranial nerves may produce the following symptoms: Nystagmus (tremor of the eyeball); strabismus; ptosis; irregular, sluggish pupils; and partial deafness or blindness.

Involvement of the Spinal Nerves.—There is extreme cutaneous hyperæsthesia, so that the slightest touch excites pain. The muscles of the extremities are stiff and may twitch, but are rarely palsied. The patellar reflex is usually diminished. The joints are occasionally red, swollen, and painful.

Febrile Symptoms.—The temperature is irregular in its course and indefinite in its duration; ordinarily it ranges be-

tween 101° and 103°, but in some cases it is almost normal, and in others it is very high. The pulse is rapid and full; the bowels are constipated; and the urine may contain albumin and sugar. Polyuria is an occasional symptom.

The Eruption.—The eruption is neither constant nor peculiar. In many cases a blotchy purpuric rash appears over the entire body. Herpes facialis is also frequently observed. In other cases urticaria, or a roseolar or erythematous rash appears.

The Blood.—Leucocytosis is always present.

Lumbar Puncture.—In a large proportion of the cases diplococci are found either on microscopic examination or in culture.

The duration is from a few hours to several weeks. In favorable cases, convalescence is very protracted.

Fulminant Form.—There is an abrupt onset with a chill, followed by vomiting, headache, moderate fever, convulsions, a petechial or purpuric rash, and death in a few hours from collapse.

Abortive Form.—The disease begins abruptly with grave symptoms, but terminates in a few days in recovery.

Intermittent Form.—The fever is characterized by intermissions or marked remissions which occur daily or every other day.

Diagnosis. *Typhoid Fever.*—The gradual onset, the regular fever, the diarrhœa and tympanites, the Widal reaction, and the absence of rigidity, of intense pain in the back and limbs, of facial palsies and of herpes, will separate typhoid from cerebro-spinal fever.

Typhus Fever.—The regular fever, the absence of intense pain in the back and limbs, of facial palsies, and of muscular rigidity, will distinguish typhus from cerebro-spinal fever.

Acute articular rheumatism may resemble cerebro-spinal meningitis, but the early involvement of the joints, the acid sweats, and the absence of rigidity, of eruption, and of facial palsies, will distinguish it from cerebro-spinal meningitis.

Tuberculous Meningitis.—In this disease the onset is less abrupt; there is less tendency to opisthotonos; herpes is rare;

and petechiæ are always absent. Tuberculous meningitis in the adult is always secondary to tuberculosis elsewhere.

Prognosis.—The mortality varies in different epidemics from 20 to 80 per cent. The prognosis should always be guarded; the mildest cases may prove fatal. Severe cerebral symptoms usually indicate a fatal termination.

Complications and Sequelæ.—Defective vision from inflammation of the cornea or retina, or from atrophy of the optic nerve; defective hearing from inflammation of the auditory nerve, or from suppurative inflammation of the internal or middle ear; pneumonia; arthritis; aphasia; peripheral palsies; imbecility; chronic hydrocephalus; and persistent headache from chronic meningitis.

Treatment.—A liquid or semi-liquid diet. Ice-bags may be applied to the head and along the spinal column. Pain and restlessness should be relived by morphine, bromides, or chloral. Morphine is especially efficacious, and may be injected along the course of the most painful nerve-trunks. Dry or wet cups over the spine are sometimes useful. Iodide of potassium (gr. v–x thrice daily) may be administered internally. Dr. Pepper recommends quinine (gr. v thrice daily) with the fluid extract of ergot (℥j every three or four hours). When the pulse weakens, stimulants should be given freely. High fever may be controlled by sponging with cold water, by the cold pack, or by the internal use of phenacetin or antipyrin.

During convalescence, iodide of potassium as an absorbent, tonics, and blisters to the spine are indicated.

MALARIAL FEVER.

(Chills and Fever, Fever and Ague, Swamp Fever.)

Definition.—A specific non-contagious disease, invariably associated with, and probably excited by, the *hæmatozoa* of Laveran, and characterized by splenic enlargement, by fever with periodic intermissions or remissions, and by a tendency to extreme anæmia.

Etiology.—A warm climate and the summer season, a moist atmosphere; low, badly-drained soil; and decaying

vegetable matter are the conditions which favor the development of the malarial poison.

Special Predisposing Causes.—Residents in the lowlands are more liable to be infected than those who dwell on the hills; one attack seems to predispose to others; visitors to malarial districts are more susceptible than permanent residents; in the night and in the early morning the air is thoroughly impregnated with the miasm, and exposure at such times is very apt to be followed by infection.

Exciting Cause.—Certain organisms belonging to the protozoa, and known as the *hæmatozoa*, are probably the exciting agents.

Manifestations.—Malarial intoxication may manifest itself, as (1) intermittent fever; (2) remittent fever; (3) pernicious malarial fever; and (4) chronic malarial cachexia.

Pathology.—Various forms of hæmatozoa are noted, some of which are distinct species, while others represent simply phases of existence in the life-history of the same organism. A small colorless amœboid body enters the red blood-corpuscle, increases in size, and becomes pigmented from the hæmoglobin of the corpuscle. When the host is destroyed the granules of pigment collect in the centre of the organism, which finally divides into a number of small hyaline bodies, each of which begins a new cycle of existence. The chills or paroxysms occur at the time of sporulation, and are doubtless due to the production of a toxine. The parasite of *tertian intermittent fever* requires forty-eight hours to complete its cycle of existence; hence, when a single group of these parasites exists in the blood paroxysms occur every other day. If, however, two groups coexist and sporulate on alternate days, a paroxysm occurs daily (*quotidian intermittent fever*). The parasites of *quartan intermittent fever* require seventy-two hours in which to develop and undergo sporulation; hence a single group of these organisms in the blood excites a chill every fourth day. When two groups coexist a chill occurs on two successive days, and is followed by a daily intermission. When three groups coexist a chill occurs every day (*quotidian intermittent fever*). The life-history within the body of the parasite of *remittent fever* is not definitely known. Its cycle of existence

occupies from twenty-four to forty-eight hours. Organisms with flagella sometimes develop from fully-grown hæmatozoa, but their significance is unknown.

Fig. 20.

Various forms of hæmatozoa.

In advanced malaria the blood shows a diminished number of red blood-corpuseles and an abundance of free pigment (melanæmia). The spleen is greatly swollen and deeply pigmented (ague-cake); the liver is moderately enlarged and pigmented. All the organs, including the brain and spinal cord, are discolored by the liberated pigment.

Intermittent Fever.

Symptoms.—The characteristic features of this form of malarial infection are: The intermittent type of fever, the enlargement of the spleen, the hæmatozoa in the blood, and the occurrence at regular intervals of paroxysms divided into three stages—cold, hot, and sweating.

Cold Stage.—Malaise; headache; great chilliness. The features are pinched; the lips are blue; the surface of the body is cold and covered with *cutis anserina* (goose-flesh), although the rectal temperature is high (104°–105°). Vomiting may occur. The chill lasts from a few minutes to an hour or two.

Hot Stage.—The surface temperature gradually rises; the skin becomes hot; the face flushed; the eyes injected; and the pulse full and rapid. The temperature in the axilla may reach 106° or 107°. The patient complains of severe pain in the head, back, and limbs, and of intense thirst. The urine is scanty and dark-colored. This stage usually lasts from one to five hours.

Sweating Stage.—The fever gradually subsides; the pain

grows less; free perspiration follows; and the patient falls to sleep, from which he awakes feeling fairly well.

Varieties.—When the paroxysms occur every day, the disease is termed quotidian intermittent; every other day, tertian intermittent; every fourth day, quartan intermittent.

Prognosis.—Always favorable. Even when no treatment is instituted the paroxysms gradually subside. Chronic malarial cachexia sometimes results from the acute disease.

Remittent Fever.

(Æstivo-autumnal Fever, Bilious Remittent Fever, Jungle Fever.)

In temperate zones remittent fever is observed chiefly in the autumn. The hæmatozoa appear at first as small round motile bodies with very little pigment in them, but soon these are replaced by ovoid or crescentic bodies containing central masses of coarse pigment.

Symptoms.—Malaise with moderate chilliness, followed by a continuous fever which daily remits. The maximum temperature ranges from 103° to 106°, and while this lasts the skin is hot, the face is flushed, the eyes are injected, the pulse is full and rapid, the urine is scanty, and the patient complains of pain in the head and limbs. Definite paroxysms may or may not be present. Delirium is sometimes noted; vomiting often occurs; and jaundice may develop from destruction of the red blood-corpuscles and liberation of their pigment. The spleen is enlarged, and an examination of the blood reveals hæmatozoa.

In some cases the symptoms resemble typhoid fever, and to these the term typho-malarial fever has been applied.

Diagnosis. *Typhoid Fever.*—The absence of diarrhœa, of tympanites, of eruption, and of a gradual rise in temperature, and the presence of hæmatozoa and of marked remissions will serve to separate remittent fever from typhoid.

Yellow Fever.—The splenic enlargement, the hæmatozoa, the multiple remissions, and the absence of bloody vomit will separate remittent from yellow fever.

Prognosis.—Favorable; the average duration is from one to two weeks.

Pernicious Malarial Fever.

(Congestive Chills, Malignant Malaria.)

Pernicious malarial fever is found chiefly in the tropics. It is invariably associated with the parasite of remittent fever. There are three varieties: algid, comatose, and hemorrhagic.

Symptoms. *Algid.*—The symptoms resemble the cold stage of cholera. The surface is cold; the temperature may be subnormal; there is great prostration; the features are pinched; the pulse is feeble. Vomiting and purging may follow; death often results in collapse.

Comatose.—There is delirium, rapidly followed by stupor and coma; the latter may or may not be associated with convulsions. The skin is hot; the face is flushed; the eyes injected; and the temperature high. The symptoms gradually disappear, but unless the patient is speedily cinchonized they return and commonly prove fatal.

Hemorrhagic.—In this form hemorrhages occur from the mucous membranes, especially from the kidneys, stomach, and bowels, and the patient is frequently jaundiced.

Diagnosis.—The algid form may resemble *cholera*, but the history, the absence of an epidemic, and the presence of the hæmatozoa in the blood will render the diagnosis apparent.

Yellow Fever.—The hemorrhagic form may resemble yellow fever, but the splenic enlargement, the late appearance of jaundice, the presence of hæmatozoa in the blood, and the absence of an epidemic will serve to distinguish the two diseases.

Prognosis.—Extremely guarded; the first paroxysm rarely kills, but unless the patient is thoroughly cinchonized a second one may prove fatal.

Chronic Malarial Cachexia.

Definition.—A chronic manifestation of malaria, characterized by anæmia, by a sallow appearance of the skin, and by splenic enlargement.

Etiology.—It may result from repeated attacks of the

acute disease, or it may develop as a primary condition from slow infection.

Symptoms.—The patient is thin and pale; the complexion is of a dirty yellow or muddy hue; fever is often absent; if present, it is slight and irregular; the spleen is considerably enlarged. There is great weakness from the attending anæmia. Headache and neuralgia are common symptoms. Hæmaturia is sometimes observed.

Diagnosis. *Leucæmia.*—The history, the absence of leucocytosis and of lymphatic enlargements, and the presence of hæmatozoa in the blood will indicate malaria.

Prognosis.—Guarded. When the spleen is very large and there is extreme anæmia, recovery rarely follows.

Other Manifestations of Malaria.

One of the following conditions may be the chief manifestation of malarial intoxication: Neuralgia, headache, hæmaturia, purpura, orchitis, or paraplegia.

Malarial infection seems to predispose to certain cases of dysentery, of pneumonia, and of amyloid degeneration of the viscera.

Treatment of Malarial Diseases. *Prophylaxis.*—Patients living in malarial districts should avoid the night and early morning air, and should take quinine (gr. iij–v a day) during the season in which the disease is prevalent.

Cold Stage of Intermittent.—Cover the patient with blankets, and apply hot cans or hot bottles to the feet. When the chill is severe and prolonged, morphine is very useful; it may be given hypodermically. Hoffmann's anodyne may be employed as a substitute. Inhalations of nitrite of amyl are followed by dilatation of the superficial bloodvessels, and in this way serve to shorten the chill.

Hot Stage of Intermittent.—Sponge the body with cool water, and if the symptoms are severe phenacetin may be given to lower the temperature and to lessen the pain.

The Interval.—It is well to begin the treatment by the administration of a laxative, and calomel may be selected. This should be followed by quinine (gr. xv–xx) in divided doses, so that the last dose is taken two hours before the time

of the expected paroxysm. In children, quinine may be given in lozenges made with chocolate and sugar. In adults, it is best administered in fresh pills or in capsules. These doses of quinine should be continued until the paroxysms disappear, when the amount may be gradually diminished. The treatment should be continued for several weeks. During convalescence it is advisable to give arsenic in the form of Fowler's solution with the quinine. The following pill is also useful in the convalescence of malaria :—

℞ Acid. arsenosi, gr. ss ;
Quinin. sulph., ʒj ;
Ferri pyrophos., gr. xxx ;
Pulv. capsici, gr. xv.—M.
Ft. in pil. No. xxx.
Sig.—One thrice daily.

Remittent Fever.—Absolute rest. A light diet. Quinine (gr. xx–xxx) should be given in divided doses in the course of a day. A laxative dose of calomel is a valuable adjunct to the antiperiodic treatment. When the stomach is irritable calomel and soda may be given by the mouth, and the quinine by the rectum or hypodermically. In some cases Warburg's tincture is useful ; half an ounce undiluted may be given, and repeated in two or three hours. After its administration the patient should be thoroughly covered with blankets so as to favor free diaphoresis.

Pernicious Malarial Fever.—From fifty to a hundred grains of quinine must be given before the second paroxysm occurs. It is advisable to begin at once without waiting for the intermission ; and twenty to thirty grains may be given hypodermically every two or three hours.

℞ Quininæ sulph., gr. xl ;
Sat. sol. acid. tartar., ♏ xlviij ;
Aquæ destil., q. s. ad fʒij.—M.
Sig.—♏ xxx = gr. x.

When the pulse weakens, stimulants, like whiskey, ammonia, and strychnine, should be employed. High temperature should be controlled by the external application of cold. In the algid form, heat should be applied externally, and opium given by the mouth or hypodermically. In the hemor-

rhagic form, opium is also useful, and it may be associated with hæmostatics like turpentine, erigeron, or hamamelis.

Chronic Malarial Cachexia.—Iron, quinine, and arsenic are the remedies indicated.

SCARLET FEVER.

(Scarlatina.)

Definition.—An acute contagious disease, characterized by high fever, a rapid pulse, a punctiform scarlet rash, sore throat, and an unusual tendency to nephritis.

Etiology.—The specific poison of scarlet fever has not been isolated. The contagium is usually carried through clothes or other fomites, or in food like milk. The disease can be transmitted by direct inoculation. The poison is tenacious and of extreme vitality; infected clothes, unused for years, have led to outbreaks. The young are especially predisposed, but not equally so. One attack does not give absolute immunity, but second attacks are uncommon.

Pathology.—The throat is inflamed and sometimes ulcerated; the liver and spleen are engorged; the muscles reveal granular degeneration. Klein has observed hyperæmia and cell-proliferation, not only in the throat and kidneys, but throughout the intestinal canal. The kidneys frequently show the lesions of hemorrhagic nephritis, the glomeruli being especially involved. The rash is rarely detected after death.

Varieties.—(1) Simple; (2) anginoid; (3) malignant.

Period of Incubation.—A few hours to a week.

Symptoms.—The disease generally begins suddenly, occasionally with a chill, but more commonly with vomiting or convulsions.

Throat Symptoms.—Pain and difficulty in swallowing; fulness and tenderness beneath the jaw; enlargement of the lymphatic glands. The tongue is at first heavily coated and red at the tip and edges; in a few days the coating almost entirely disappears, and the papillæ become bright red and swollen. This appearance has given rise to the term "strawberry tongue." The pillars, tonsils, uvula, and pharyngeal vault are deeply injected and may reveal a punctiform efflo-

rescence before the rash develops on the skin. In severe cases the tonsils may be the seat of follicular inflammation, or may be covered with false membrane.

Eruption.—A scarlet-red punctiform rash appears at the end of the first, or at the beginning of the second day, on the neck and chest, and rapidly spreads over the entire body. It disappears on pressure, a white line remaining for a second or two when the finger-nail is drawn through it. It may be uniform or it may occur in discrete patches surrounded by healthy skin. In five or six days the red color gradually fades and scaly desquamation soon follows.

In some cases the rash is pale and scarcely visible, in others it is slightly papular or vesicular (scarlatina miliaris); in malignant cases it may be petechial.

Febrile Symptoms.—The fever rises abruptly, reaching its maximum (104°–105°) in twenty-four or forty-eight hours, remains nearly uniform for three or four days, and then falls by lysis. The duration of the febrile period is from seven to nine days. The pulse is very rapid,—out of proportion to the fever; the respirations are hurried; the appetite is lost; the bowels are constipated; and the urine is scanty, high-colored, and often contains albumin.

Nervous Symptoms.—Restlessness, headache, insomnia, delirium, and convulsions may occur in the course of the disease. Convulsions developing late in the disease are very significant of uræmia.

Anginoid Scarlet Fever.—This form is characterized by severe throat symptoms. The tonsils are much swollen and are often covered with false membrane. The fever is high and the prostration is profound. Ulceration of the throat frequently occurs. Death may result from exhaustion, aspiration-pneumonia, or from hemorrhage due to ulceration of the carotid artery.

Malignant Scarlet Fever.—The onset is abrupt, with a chill, vomiting, or convulsion; the fever is very high (106°–107°); the pulse is rapid and feeble; delirium sets in, and is followed by coma. Death may result before the appearance of the rash, in twenty-four or forty-eight hours.

Complications. *Nephritis.*—This usually develops during

convalescence, and as it may be unassociated with subjective symptoms the urine should be examined daily in order to detect its presence; in other cases its advent is recognized by the suppression of urine, by uræmia, or by dropsy. Nephritis may be the immediate cause of death, but more commonly it ends in recovery; it sometimes leads to chronic renal disease.

Among other complications may be mentioned hyperpyrexia, endocarditis, pericarditis, pneumonia, suppuration of the lymphatic glands, ophthalmia, inflammation of the middle ear, chorea, and a peculiar inflammation of the joints resembling rheumatism.

Diagnosis.—*Acute Tonsillitis* may resemble scarlet fever, especially when the former is associated with an erythematous rash; but in tonsillitis there is no history of contagion, the pulse is proportionate to the fever; the rash, if present, is not punctiform; the tongue has not the strawberry appearance; and there is no tendency to nephritis.

Diphtheria.—The onset is less abrupt; there is more prostration; false membrane, containing the Klebs-Löffler bacillus, is always present; a cutaneous rash is usually absent; and the tongue does not present a strawberry appearance.

Measles.—The sore throat is less marked; catarrhal symptoms are present; the rash appears later, is papular, and forms in crescentic-shaped patches; the fever shows a decided remission on the second or third day; and the pulse is proportionate to the fever.

Rötheln.—This may be difficult to distinguish from scarlatina, but the fever is not so high, nor the pulse so rapid; the post-cervical glands are more swollen; there is no tendency to nephritis; and the rash is not punctiform.

Accidental Rashes.—Certain drugs like belladonna, quinine, and copaiba, and certain foods, like crabs and oysters, may produce a rash like that of scarlet fever, but it is not punctiform, and is not associated with high fever, sore throat, and rapid pulse.

Prognosis.—Always guarded. The mortality varies in different epidemics from 5 to 40 per cent.

Treatment. — Isolation. Absolute rest. Liquid diet. The surface of the body should be anointed two or three times

daily with cold cream, cocoa-butter, or carbolized vaseline. The patient should be encouraged to drink water or lemonade freely. Gastric irritability may call for small doses of calomel, bismuth, or nitrate of silver. When the stomach is retentive, the tincture of the chloride of iron may be given with small doses of dilute hydrochloric acid, thus:—

℞. Tinct. ferri chlor., f℥ij;
Acid. hydrochlor. dil., f℥j;
Syr. limonis, f℥j;
Aquæ, q. s. ad f℥iij.—M.
Sig.—Teaspoonful in water every two or three hours.

The fauces and pharynx should be kept clean by antiseptic washes or sprays, such as Dobell's solution, dilute peroxide of hydrogen, or dilute listerine.

Cerebral symptoms may be controlled by bromide of potassium, chloral, by an ice-bag to the head, or, when due to fever, by the cold bath.

High fever is best treated by sponging, by the cold pack, or by the graduated cold bath.

The urine should be examined daily for evidence of nephritis, and, if the latter arises, the diet should be cut down to skimmed milk or buttermilk; dry cups may be applied to the loins; the bowels kept active by Epsom or Rochelle salt; and diaphoresis encouraged by small doses of jaborandi.

Cardiac weakness will call for stimulants like alcohol, ammonia, strychnine, and digitalis.

Convalescence should be guarded and protracted.

MEASLES.

(Rubeola, Morbilli.)

Definition.—An acute contagious disease, characterized by catarrh of the respiratory tract, moderate fever, and a red papular eruption, which appears on the fourth day and terminates in two or three days by branny desquamation.

Etiology.—Measles is highly contagious, and the poison may be transmitted through clothes and other fomites. The contagium is apparently associated with the nasal and bronchial secretion, but it has not been isolated. It is most

commonly observed in children, but unprotected adults are very liable to be attacked. It is essentially an epidemic disease, but now and then sporadic cases occur. One attack is fairly protective, but does not give absolute immunity.

Pathology.—The lesions consist in catarrh of the entire respiratory tract. Gastro-intestinal catarrh is not uncommon. In fatal cases such complications as capillary bronchitis, catarrhal pneumonia, and pulmonary collapse are frequently observed.

Period of Incubation.—About two weeks.

Symptoms. *Prodromes.*—Chilliness, coryza, watering of the eyes, photophobia, cough, and drowsiness.

The Fever.—The temperature rises rapidly to 102° or 103°, but on the second day there is a decided remission which continues until the fourth day, when the eruption appears; at this time it again rapidly runs up to, or beyond, its original height where it remains for two or three days and then falls by crisis.

The Catarrh.—Redness of the conjunctivæ, lachrymation, sneezing, hoarseness, cough, and expectoration. There may be vomiting or diarrhœa.

The Eruption.—This appears about the fourth day on the face, and rapidly spreads over the entire body. It is composed of small, dark-red, velvety papules, which form groups having crescentic borders. In two or three days the eruption begins to fade, and branny desquamation soon follows.

Minute bluish-white specks surrounded by a red areola may be seen on the mucous membrane of the cheeks and lips one or two days before the skin eruption appears (Koplik's sign).

Malignant, or Hemorrhagic Measles.—This form occurs under bad hygienic conditions, and is characterized by a petechial rash, by hemorrhages from the mucous membranes, and by profound prostration.

Complications and Sequelæ. — Capillary bronchitis, catarrhal pneumonia, tuberculosis, otitis, gastro-intestinal catarrh, cancrum oris, and paralysis.

Diagnosis. *Rötheln.*—Prodromes are often absent; fever and catarrh are slight; sore throat is marked. The rash

appears on the first or second day as a diffuse red blush, or as small pale-red spots which do not form crescentic-shaped patches; desquamation is scarcely noticeable.

Scarlet Fever.—The fever is high and lacks the pre-eruptive remission; sore throat is present instead of general catarrh; the eruption appears on the first or second day as a diffuse punctiform rash; the pulse is out of proportion to the fever; and there is much greater tendency to nephritis.

Prognosis.—Guardedly favorable. Complications are apt to occur and render the prognosis grave.

Treatment.—Isolation. A darkened well-ventilated room; absolute rest. A liquid diet. Such refrigerant remedies as sweet spirits of nitre and liquor ammoniæ acetatis are indicated and may be combined with a little aconite.

℞ Spt. æther. nitrosi, f℥j;
Liq. ammon. acetatis, q. s. ad f℥iij.—M.
Sig.—A teaspoonful every two hours.

When the bronchitis is severe it is advisable to envelop the chest with a cotton jacket, and to administer expectorants with sedatives like paregoric.

℞ Liq. potass. citrat., f℥iss;
Tinct. opii camph., f℥iij;
Syr. ipecac., f℥ij;
Syr. acaciæ, f℥ss;
Aquæ, q. s. ad f℥iij.—M.
Sig.—A dessertspoonful every two hours for a child of five years.

Gastric irritability should be relieved by small doses of bismuth or by calomel and soda. During desquamation the skin should be anointed two or three times daily. High fever is best controlled by sponging with tepid water. During convalescence nutrients like cod-liver oil and malt, and tonics like iron, quinine, and strychnine are indicated.

RÖTHELN.

(Rubella, German Measles, Epidemic Roseola.)

Definition.—An acute contagious disease resembling both scarlet fever and measles, but differing from these in its short course, slight fever, and freedom from sequelæ.

Etiology.—The disease is highly contagious, and the poison may be carried on clothes or other fomites. It gener-

ally occurs in epidemics, but sporadic cases are not uncommon. It is most frequently observed in children, but unprotected adults are not exempt. One attack usually protects from another, but not from measles or scarlet fever.

Period of Incubation.—About two weeks.

Symptoms.—Prodromes are slight, or altogether absent. The disease begins with drowsiness, slight fever, and sore throat. The eruption appears on the first or second day, and varies considerably in its character. In some cases the rash is composed of pale-red, scarcely elevated papules, which are more or less discrete (*rubella morbilliforme*); in others the rash is bright red and diffuse like that of scarlet fever (*rubella scarlatiniforme*). It begins on the face and rapidly spreads over the entire body, but it fades so rapidly that the face may be clear before the extremities are affected. Slight desquamation frequently follows, though it is often absent. Apart from the sore throat, the catarrhal symptoms are slight. The superficial cervical and posterior auricular glands are more swollen than in measles.

The duration is from three to five days.

Prognosis.—Good. Complications are rare.

Treatment.—Rest. Liquid diet. Refrigerants. Sponging with tepid water.

SMALLPOX.

(Variola.)

Definition.—An acute contagious disease, characterized by vomiting; lumbar pains; an eruption which is at first papular, then vesicular, and finally pustular; and by fever which is marked by a distinct remission beginning with the advent of the eruption, and lasting until the latter becomes pustular.

Etiology.—The poison of smallpox is extremely tenacious; it may remain latent in clothes or other fomites for a long time, and then be capable of exciting the disease. The virulent principle is doubtless contained in the pustules and in all the excretions of the body, but it has not been isolated. Unless protected by vaccination or a previous attack, nearly every one is susceptible, from the aged to the child *in utero*. The colored race seem especially predisposed.

Pathology.—The eruption consists in an infiltration of cells into the *rete mucosum* or into the true skin. The cells ultimately undergo liquefaction-necrosis, when suppuration soon follows. Genuine pocks are frequently found in the mouth, œsophagus, and larynx, and rarely in the stomach, trachea, and bronchi. The spleen is engorged. The organs and muscles reveal fatty and parenchymatous degeneration.

Varieties.—Discrete; confluent; malignant; varioloid.

Fig. 21.

Temperature Curve in Smallpox.

Symptoms. **Discrete Smallpox.** — The disease usually begins with a chill or series of chills, followed by vomiting and intense lumbar pains. The fever rises rapidly, reaching its maximum (104°–105°) in forty-eight hours, and continues high until the third or fourth day, when it falls several degrees; this remission lasts until the seventh or eighth day,—that is, the time of pustulation,—when it again rises. The secondary or suppurative fever shows marked fluctuations; its height is proportionate to the number of pustules; and it falls by lysis about the eighteenth day of the disease. The pulse is full and rapid (120–140); the breathing is hurried; the skin is dry; the bowels are usually constipated, though diarrhœa is not uncommon; and the urine is scanty and frequently albuminous.

The Eruption.—About the third or fourth day small red spots are noticed on the forehead, face, and wrists; these are rapidly converted into smooth round papules which feel like shot under the skin. The eruption rapidly spreads over the entire body. About the third day the papules are converted into clear vesicles, which present a depression or umbilication

at their summit. They are also loculated, *i. e.* divided into compartments by fibrinous partitions, so that when pricked with a needle all of the contained fluid does not escape. In two or three days the clear fluid becomes turbid and the vesicles are gradually converted into pustules. The latter soon lose the umbilicated appearance. Between the lesions the skin is œdematous, so that the body is swollen and the features are unrecognizable. In three days more the pustules dry up, or break and form soft yellow crusts which exhale a peculiar, offensive odor; they adhere to the skin for a week or more. When the scabs fall off, scars, or pock-marks generally remain, constituting a permanent deformity.

At the beginning of the disease, before the true variolous eruption appears, either a red blush or a macular rash is often observed on the inner side of the arms and thighs.

Confluent Smallpox.—The papules are abundant, and soon coalesce. The extremities are swollen and painful. The secondary fever is very high and irregular. True pocks nearly always develop in the air-passages and give rise to a copious fetid discharge from the nose and throat, to hoarseness, and to cough. Delirium, stupor, and subsultus are frequent symptoms. If the patient recovers, it is after a tedious convalescence, with great facial disfigurement, and often with defective vision and hearing.

Malignant Smallpox.—In some cases the disease is ushered in with high fever, lumbar pains, and great prostration. Soon ecchymoses appear on the skin; bleeding from the mucous membranes follows; and death results before a true variolous rash appears. In other cases the disease advances like ordinary smallpox up to the pustular stage; then the pustules become effused with blood, and bleeding from the mucous membrane follows. This form is also very fatal.

Varioloid.—This is modified smallpox occurring in one who has been partially protected by previous vaccination. The symptoms are mild; the eruption resembles that of common smallpox, but is usually very scant; secondary fever is absent.

Complications and Sequelæ.—Broncho-pneumonia;

pleurisy; inflammations of the eye (keratitis, iritis, conjunctivitis); otitis; arthritis; and boils.

DIAGNOSIS. *Varicella.*—The symptoms are milder; prodromes are generally absent; the eruption appears earlier, is more superficial, lacks an inflammatory areola, is more abundant on the trunk than the face, and is rarely umbilicated.

Secondary Syphilis.—The history; the absence of fever; the symmetrical distribution of the eruption; its dark-coppery color; its polymorphous character (papules, vesicles, and pustules associated in a limited area); and the absence of itching will indicate syphilis.

PROGNOSIS.—This depends upon the virulence of the epidemic, the degree of protection by vaccination, and the amount of the eruption. In discrete cases, it is generally favorable; in the confluent, grave; in the malignant, almost hopeless.

TREATMENT.—The *prophylactic treatment* consists in vaccination.

The Attack.—Isolation. Every precaution must be taken to prevent the spread of the disease. The other members of the family should be vaccinated at once. The room should be cool and well ventilated. The diet must be liquid or semi-liquid, and may consist of milk, meat broths, eggs, etc. The free use of water, lemonade, or soda-water should be encouraged. The intense lumbar pains should be relieved by opium and the application of hot-water bags. Gastric irritability may call for bismuth or calomel and soda. The naso-pharynx should be kept clean by antiseptic washes and sprays, and Dobell's solution, dilute listerine, or dilute peroxide of hydrogen may be used for this purpose. The eyes must be kept clean by being washed several times a day with a saturated solution of boric acid. Stimulants are often indicated. High fever may be controlled by antipyrin or phenacetin, or by the cold pack or cold bath.

The prevention of Pitting.—The room should be darkened, and the exposed parts covered with cloths soaked in dilute carbolic acid or bichloride of mercury, or with masks upon which has been spread some simple ointment, as one of mercury or of zinc. Unfortunately, when the lesions are deeply situated there is no means of preventing pitting. The separation of the scabs may be facilitated by the use of warm baths.

VARICELLA.

(Chicken-pox.)

DEFINITION.—An acute contagious disease of short duration, characterized by slight fever and a discrete vesicular eruption, which disappears in two or three days by desiccation.

ETIOLOGY.—The disease occurs sporadically and epidemically. It is observed chiefly in children, but adults are not exempt. One attack usually protects from others. It bears no relation to smallpox.

PERIOD OF INCUBATION.—One to two weeks.

SYMPTOMS.—Slight fever and the appearance of a vesicular eruption within the first twenty-four hours. The vesicles appear in crops over two or three days; they are superficial, rarely umbilicated, and lack the red areola which is seen around the vesicles of variola. They rarely become pustular, and are only occasionally followed by scars. The duration is about a week. In rare instances gangrene occurs around the vesicles or in other parts (varicella gangrenosa).

DIAGONSIS. *Smallpox.*—The slight fever; the absence of lumbar pains; the early appearance of the vesicles, their marked variation in size, and their greater intensity on the trunk; and the absence of umbilication and red areola will serve to distinguish varicella from smallpox.

PROGNOSIS.—Always favorable.

TREATMENT.—Rest in bed. A light diet. The application of some sedative lotion or ointment to allay itching and to prevent scratching.

VACCINIA.

(Vaccination, Cow-pox.)

DEFINITION.—A general disease with a local manifestation resembling the pock of variola, and acquired by inoculation with the virus of cow-pox.

HISTORY AND OBJECT.—The value of vaccination as a means of protection against smallpox was first made known to the world in a paper published by Edward Jenner in 1798.

Recent vaccination gives almost complete immunity from variola; the mortality of smallpox acquired after vaccination is almost inversely proportionate to the number of true vaccine scars.

Etiology.—Vaccinia is induced by inoculating the arm with fresh virus obtained from the udder of a calf suffering from cow-pox (bovine virus), or from the vesicle of a patient who has already been vaccinated (humanized virus). The former is preferable on account of the readiness with which the fresh article can be obtained, and on account of its freedom from other poisons, like syphilis. It has been shown that the addition of glycerin to vaccine lymph serves to preserve it, and to free it from pathogenic bacteria.

Time of Performance.—The first vaccination should be made about the third month, the second at the seventh year, and the third at puberty. It should always be repeated when smallpox is prevalent.

Performance of Vaccination.—The arm should be rendered aseptic, and the skin scratched with a lancet or with the ivory point containing the lymph until red serum begins to ooze, when the moistened virus should be carefully worked in. The spot must be carefully protected from the clothes until thoroughly dry.

Symptoms.—About the second or third day after the operation a papule surrounded by a red areola forms at the seat of inoculation. In two or three days the papule is converted into a clear vesicle, which is umbilicated at its summit; the surrounding tissues are red, tender, and considerably infiltrated. About the seventh or eighth day the vesicle is perfected and its contents become turbid; this lasts until the twelfth day, when it dries up and forms a scab, which separates during the third week and leaves behind a pitted scar. During the course of the eruption there are slight fever, malaise, restlessness, and enlargement of the axillary glands.

Complications.—Erysipelas, abscess, and various cutaneous eruptions. Syphilis has occasionally been transmitted through humanized virus.

ERYSIPELAS.

(St. Anthony's Fire.)

DEFINITION. — An acute contagious disease excited by streptococci, and characterized by a peculiar inflammation of the skin and subcutaneous tissue, irregular fever, and a tendency to relapse.

ETIOLOGY.—The disease is somewhat contagious and the poison can be carried in fomites. Certain families and certain individuals seem particularly predisposed. Puerperal women and wounded persons are very susceptible. Diseases which lower the vitality, especially Bright's disease, predispose. One attack does not protect against a recurrence, but rather favors it. Erysipelas was formerly divided into traumatic and idiopathic varieties; but the two are identical, and it is probable that in those cases in which there is no conspicuous wound there is a slight abrasion through which the poison gains admittance.

The *exciting cause* is doubtless the streptococcus pyogenes.

PATHOLOGY.—Erysipelas most frequently manifests itself on the face. The part is bright red in color, swollen, indurated, and sharply circumscribed. The various strata of the skin are infiltrated with serum, and leucocytes and streptococci are found in the lymph-spaces. In severe cases the inflammatory products are converted into pus, and abscesses form.

PERIOD OF INCUBATION.—Three to seven days.

SYMPTOMS.—Prodromes are sometimes present, and consist of slight fever, chilliness, malaise, tingling of the part to be affected, and sometimes enlargement of neighboring lymphatic glands. In many cases the disease is ushered in suddenly with a chill, followed by pain in the head and limbs and a high, irregular fever. The temperature may reach 103° or 104° in twelve or twenty-four hours. The pulse is full and rapid; the tongue is heavily coated; the appetite is lost; the bowels are constipated; and the urine is scanty and often slightly albuminous.

Local Phenomena.—The inflammation usually begins in the neighborhood of the nose, and spreads upward and laterally over

the head to the neck, where it frequently stops. The affected part has a crimson hue; it is swollen and tense, and frequently ends in a sharply-defined ridge, beyond which, however, projections can be felt advancing into the subcutaneous tissue. The surface of the inflamed patch is at first smooth and glazed, but later it is covered with minute vesicles or blebs. The patient complains of burning and tingling; the surrounding parts are extremely œdematous, so that the features may be scarcely recognizable. In four or five days the redness begins to fade and the swelling to subside; desquamation follows; the general symptoms improve; and the fever falls by crisis. The average duration is from a week to ten days. Relapses are extremely common.

Erysipelas Ambulans.—Sometimes the inflammation disappears in one place and reappears in another, and so continues indefinitely. In such cases typhoid symptoms, such as muttering delirium, a brown, fissured tongue, and subsultus tendinum, develop.

Complications. — Inflammation of serous membranes (pericarditis, pleuritis, meningitis), œdema of the larynx, nephritis, hyperpyrexia, ulcerative endocarditis, and septicæmia.

Diagnosis. *Erythema.* — The absence of high fever, of marked swelling, and of an abrupt ridge will serve to distinguish erythema from erysipelas.

Acute Eczema.—The swelling is less marked; the itching is intense; the swel'ing and redness are not circumscribed, but shade gradually into healthy tissue; and there is no fever.

Prognosis.—In the robust the prognosis is favorable. In the old, in alcoholic subjects, and in those suffering from chronic nephritis, the prognosis must be guarded. Ambulatory erysipelas may kill by exhaustion.

Treatment.—Isolation; absolute rest; a nutritious diet. It is well to begin the treatment with a saline or mercurial laxative. The tincture of the chloride of iron seems to exert a beneficial influence; it may be given in doses of twenty drops every two hours. Quinine (gr. v thrice daily) is also useful. When there is much restlessness and insomnia, bromide of potassium, chloral, or opium may be administered.

Local Treatment.—One of the following applications may be

employed: Cloths wrung out in a solution of bichloride of mercury (1–5000), or in a saturated solution of boric acid, or in lead-water and laudanum; a dusting powder of starch and oxide of zinc; or an ointment of ichthyol.

℞ Plumbi acetatis, ʒj;
Tinct. opii, f ʒj;
Aquæ, q. s. Oj.—M.
Sig.—Shake well and apply on lint.

Or—

℞ Ichthyol, ℥ss;
Vaselin., ʒij.—M.
Sig.—Spread thickly on lint and apply to the affected part.

The injection of antiseptic remedies around the inflammatory patch, with the view of preventing its spread, is very painful and seldom efficacious.

YELLOW FEVER.

Definition.—An acute infectious disease, characterized by jaundice, epigastric tenderness, vomiting, hemorrhages, and a febrile course consisting of two paroxysms.

Etiology.—A hot climate and a warm season, salt water, bad drainage, and overcrowding favor the development of epidemics. The disease is not distinctly contagious; the poison probably undergoes some changes outside of the body, and is carried through the atmosphere, clothes, or other fomites. The colored race are less susceptible than the white. Strangers in an infected district are more liable to be attacked than residents. One attack usually confers immunity from others. The exciting cause is probably the bacillus icteroidis, a fine, motile, ciliated rod, recently described by Sanarelli.

Pathology.—The tissues are stained yellow by disintegrated blood (hæmatogenous jaundice). The liver is pale and is the seat of extensive fatty degeneration. The gastric mucous membrane is swollen, congested, and frequently ecchymosed. The spleen is not enlarged. The heart is pale and flabby. The kidneys are generally the seat of parenchymatous inflammation.

Period of Incubation.—A few hours to a week.

Symptoms. *First Stage.*—The disease begins with a chill, followed by pain in the head, back, and limbs. The temperature rises rapidly until it reaches its maximum (103°–105°). The pulse, however, is not proportionately rapid, and often remains at 70 or 80 per minute. The face is flushed, slightly icteroid, the conjunctivæ are injected, and the pupils small; the tongue is coated, the epigastrium is tender, the stomach is irritable and unretentive; the bowels are constipated; and the urine is scanty and albuminous. This stage lasts from a few hours to several days, and is followed by a marked fall in the temperature and an improvement in the general symptoms (stage of remission). At this time convalescence may begin, or the patient may pass into the second febrile paroxysm.

Second Stage.—The fever rises to its original height; the skin becomes yellow; vomiting is persistent, and the ejected material may contain dark blood ("black vomit"). Hemorrhages sometimes occur from other mucous membranes. The pulse is rapid, though not proportionate to the fever. The urine becomes very scanty and contains albumin and casts. The mind often remains clear until near the close. Death frequently results from exhaustion or uræmia, though recovery may follow the gravest symptoms.

Duration.—From a few hours to a week.

Diagnosis. *Dengue.*—This disease does not exhibit a slow pulse with the rising temperature, albuminuria, jaundice, or black vomit.

Acute Yellow Atrophy of the Liver.—The rapid pulse, the diminution in the size of the liver, the slight fever, the marked cerebral symptoms, and the presence of leucin and tyrosin in the urine will indicate acute yellow atrophy.

Remittent Fever.—This may be distinguished by the enlargement of the spleen, the multiple remissions, the presence in the blood of hæmatozoa of Laveran, and by the absence of black vomit.

Prognosis.—Always grave. The average mortality in different epidemics is from twenty to seventy per cent. In

individual cases, high fever, severe cerebral symptoms, black vomit, and suppression of urine are unfavorable features.

TREATMENT.—Absolute rest. A cool, well-ventilated room. A liquid diet. The pains in the back and limbs may be relieved by hot-water bags and the administration of morphine. For the gastric irritability a mustard plaster may be applied to the epigastrium, and cracked ice, iced champagne, carbolic acid, or small doses of calomel may be given internally. Stimulants are frequently indicated. Quinine may be given by the rectum. High fever is best controlled by the external application of cold. The black vomit results from blood-dyscrasia, and while such remedies as gallic acid, Monsel's solution, ergot, and turpentine are recommended, they usually prove useless.

ACUTE GENERAL TUBERCULOSIS.

(Acute Miliary Tuberculosis.)

DEFINITION.—An acute infectious disease excited by the tubercle bacillus, and characterized anatomically by the simultaneous formation of miliary tubercles in many parts of the body.

ETIOLOGY.—The disease usually develops in early adult life. Certain infectious diseases like measles, whooping-cough, and typhoid fever seem to predispose. General tuberculosis is almost always secondary to local tuberculosis—pulmonary phthisis or a scrofulous lymphatic gland. The bacilli are probably disseminated by the veins.

PATHOLOGY.—All the organs may be uniformly infiltrated with discrete tubercles, but more commonly certain organs, like the brain and lungs, are more affected than others.

SYMPTOMS.—Debility; loss of flesh and strength; fever moderately high (102°–104°), irregular, and marked by evening exacerbations and morning remissions; cough; hurried respirations; a brown, fissured tongue; a weak, rapid pulse; enlargement of the spleen; delirium; subsultus tendinum; and stupor.

Tubercle bacilli are rarely found in the expectoration or in the blood.

The duration is from two to four weeks.

When the lungs are chiefly affected there are: Dyspnœa, marked cough, muco-purulent and bloody expectoration, cyanosis, sibilant and subcrepitant râles, and perhaps areas over which bronchial breathing is detected.

When the meninges are chiefly affected there are: Intense headache, convulsive seizures, photophobia, delirium, facial palsies, stupor, coma, and Cheyne-Stokes breathing. Tubercles may be detected on the retina.

When the intestines and peritoneum are affected there are: Pain, tenderness, abdominal distention, and diarrhœa.

Diagnosis.—The disease closely resembles *typhoid fever*, and there is no doubt that the mortality of the latter is enhanced by included cases of unsuspected general tuberculosis. The following table will indicate the points of distinction:—

Typhoid Fever.	Acute General Tuberculosis.
Epistaxis common.	Infrequent.
The temperature rises gradually, and runs a regular course.	The temperature usually rises abruptly, and runs a very irregular course.
Diarrhœa is frequent.	Infrequent.
An eruption is generally present.	Rarely present.
No tubercles on the retina.	Occasionally detected.
Respirations are hurried.	Still more hurried.
Facial palsies are rare.	Common.
Widal reaction is present.	Is absent.

Prognosis.—Always fatal.

Treatment.—Palliative. The diet should consist of milk, eggs, and broths. Stimulants are indicated. High fever should be controlled by antipyrin or by the external application of cold.

DIPHTHERIA.

(Diphtheritis, Malignant Sore Throat, Cynanche Contagiosa.)

Definition.—An acute contagious disease excited by the Klebs-Löfler bacillus, and characterized by moderate fever,

glandular enlargements, great prostration, and a fibrinous exudation which is usually located in the throat.

Etiology.—Childhood (between three and six), defective drainage, and catarrhal conditions of the throat are predisposing factors. The poison is contained in the secretions of the throat, and may be transmitted through the atmosphere or through fomites. One attack does not protect from another, but rather predisposes.

The exciting cause is the Klebs-Löfler bacillus, which is found only in the membranous exudation. The constitutional symptoms result from the poison generated by the bacillus.

Pathology.—The false membrane is usually found on the tonsils, pillars, and pharynx, but it may extend to the mouth, larynx, or nose. The bacillus coming in contact with the throat leads to the death of the superficial cells, which ultimately undergo coagulation-necrosis. The irritation causes a migration of leucocytes, and these undergo a similar necrosis. The membrane thus formed is of a grayish-white color, and is more or less adherent, so that when torn off it leaves a raw surface. Sometimes the necrosis extends to the deeper tissues and causes widespread ulceration and even gangrene. Microscopically, the pseudo-membrane is composed of fibrin, leucocytes, bacteria, and the remains of epithelial cells. The lymphatic glands are considerably swollen. The spleen is engorged. The various organs and the muscles reveal fatty and parenchymatous degeneration. Examination of the lungs frequently shows capillary bronchitis, catarrhal pneumonia, and collapse.

In some cases the blood is dark and fluid, while in others firm clots are found within the heart.

Types.—Diphtheria may be divided according to the location of the exudate into: (1) Faucial; (2) laryngeal; (3) nasal; (4) cutaneous. According to the severity of the attack it may be divided into: (1) Mild; (2) grave; (3) malignant.

Period of Incubation.—Two to ten days.

Symptoms. *Faucial Diphtheria.*—The disease commonly begins with chills, moderate fever, malaise, and sore throat. The fever, as a rule, is not very high (102°–104°) and its course is quite irregular. The pulse soon becomes rapid and

feeble; the bowels are constipated; the urine is scanty and frequently albuminous; and the prostration and pallor are often out of all proportion to the severity of the febrile symptoms.

Local Phenomena.—The child complains of difficult swallowing; the muscles of the neck feel stiff; there is tenderness under the jaw; the lymphatic glands are considerably swollen; and the fauces are covered with a grayish-white membrane which when stripped off leaves a raw bleeding surface, and is soon followed by a similar deposit. The membrane may spread to the nose or larynx.

The course of the disease is indefinite, the average duration being from one to two weeks.

Laryngeal Diphtheria.—This is usually secondary by extension from the fauces, but it is occasionally primary. It is recognized by hoarseness or aphonia, croupy cough, progressive dyspnœa, and stridulous breathing. The alæ of the nose play; the sterno-cleido-mastoids are prominent; the supra-sternal notch is deepened; and the base of the chest is retracted. Shreds of false membrane are sometimes expectorated in the violent fits of coughing. The febrile symptoms are usually slight. Death often results from suffocation, but recovery is not impossible in the most unpromising cases.

Nasal Diphtheria.—This is nearly always secondary. It is recognized by grave constitutional symptoms—high fever, marked glandular involvement, and great prostration; by an offensive discharge from the nose; by epistaxis; and by excoriation of the lips. The false membrane may be detected on inspection.

Cutaneous Diphtheria.—This form may be primary or secondary. The constitutional symptoms are similar to those of faucial diphtheria.

Complications and Sequelæ.—Capillary bronchitis, catarrhal pneumonia, pulmonary collapse, myocarditis, otitis media, nephritis, and paralysis.

Diphtheritic Paralysis.—This generally occurs during convalescence and is observed in about fifteen per cent. of all cases. There is no relation between the severity of the attack of diphtheria and the liability to paralysis; mild cases, which are thought to be simple pharyngitis, are sometimes followed by

troublesome paralysis. The pharynx is the most common seat, and the palsy is recognized by difficult swallowing and the regurgitation of liquids through the nose. Next in frequency the eyes are involved, and strabismus or ptosis develops. The heart may be affected, and if sudden death does not result, the condition may be manifested by a remarkable slowing of the pulse. In some instances there is an extensive involvement of the extremities. The paralysis is due to a toxic neuritis.

Diagnosis. *Scarlet Fever.*—The onset is more sudden; the fever is higher; the pulse more rapid; the tongue presents a strawberry appearance; a red punctiform rash appears on the first or second day; and if membrane appears on the throat, it does not contain the Klebs-Löffler bacillus.

Follicular Tonsillitis.—In this disease the yellow patches are *in* the gland, not *on* it. If false membrane is present, it is confined to the tonsils and does not contain the Klebs-Löffler bacillus. Albuminuria is very rarely present.

Prognosis.—Always guarded. The mortality varies in different epidemics from 10 to 50 per cent. When the constitutional symptoms are mild, and the membrane is confined to the fauces and shows little tendency to spread, the prognosis is quite favorable. The nasal and laryngeal forms are always very grave.

Treatment. *Prophylaxis.*—As diphtheria is prone to attack unhealthy mucous membrane, naso-pharyngeal catarrh in children should receive careful attention. Large tonsils and adenoid growths should be removed. Those who have been exposed to the contagion should receive immunizing doses of antitoxin. Patients with diphtheria should be kept isolated until their throats are free from virulent bacilli. The bed-room, bedding, clothing, and all utensils used by the sick should be thoroughly disinfected.

Treatment of the Attack.—Isolation; absolute rest; liquid diet. Upon the first evidence of heart-failure, stimulants, such as alcohol, strychnine, strophanthus, or caffeine, should be administered. Of the special remedies to be exhibited by the mouth, iron and mercury have the most advocates. The bichloride of mercury is well borne, and may be given in

doses of $\frac{1}{40}$ to $\frac{1}{20}$ of a grain to a child of four years. The tincture of the chloride of iron should be given, well diluted, at short intervals.

℞ Tinct. ferri chloridi, f℥ij;
Glycerini, f℥vj;
Aquæ, q. s. ad f℥iv.—M.
Sig.—Teaspoonful every hour for a child of four years.

It has been fully demonstrated that antitoxin, or the serum of immunized animals, is the best therapeutic agent in diphtheria. The investigation conducted by the American Pediatric Society has shown that the mortality under the serum treatment in 5794 cases was only 12.3 per cent., and that when the treatment was instituted during the first three days the mortality was only 7.3 per cent. Fifty per cent. of the laryngeal cases recovered without operation, and among those in which intubation was performed the mortality was 25.9 per cent., or less than half as great as has ever been reported under any other form of treatment. The strength of the serum is measured in units, the latter being equal to 1 c.cm. of "normal serum," which is the blood serum of an immunized animal so active that $\frac{1}{10}$ of a c.cm. will antagonize ten times the minimum of diphtheria poison fatal to a guinea-pig weighing 300 grams. To a child of two years 1000 units should be administered at once. On the following day, if no improvement results, 1500 to 2000 units should be administered, and repeated on the third day if necessary. Severe cases, especially when seen late, should receive the maximum dose at the outset. The prophylactic dose is 600 to 1000 units. The injections may be made in the buttocks, flanks, or subscapular region.

The atmosphere of the room should be rendered moist by slacking lime, by evaporating water on the stove or over a spirit-lamp, or by means of a steam atomizer. The addition of turpentine or of oil of eucalyptus to the water is often recommended. Iodine, or an ointment of mercury, belladonna, or ichthyol, may be applied to the swollen and tender glands. The naso-pharynx should be kept clean by antiseptic sprays or douches, and one of the following may be selected for this

purpose: Dobell's solution, dilute listerine, dilute peroxide of hydrogen, chlorine-water, or corrosive sublimate (1 : 2000).

Many solvents have been recommended; those most commonly employed are dilute lactic acid, dilute hydrochloric acid with pepsin, a solution of papayotin, and peroxide of hydrogen. The last is often useful, but it is essential that it should be fresh. When the throat is not too sensitive it may be employed undiluted. Löffler's solution is very satisfactory. The formula is—

℞ Menthol., ʒiiss;
Toluol., q. s. ad fʒx;
Solve et adde—
Alcohol. absolut., fʒij;
Liquor. ferri chloridi, fʒj.—M.
Sig.—Apply with a cotton swab.

In laryngeal diphtheria, when these means fail, tracheotomy or intubation must be resorted to.

WHOOPING-COUGH.

(Pertussis.)

Definition.—An infectious disease, characterized by catarrh of the respiratory tract and peculiar paroxysms of cough ending in prolonged crowing or whooping inspiration.

Etiology.—The disease occurs both sporadically and epidemically. It is most frequently met with in children, but unprotected adults are not exempt. The disease is unquestionably contagious, and the virus seems to be associated with the sputum. One attack protects from others.

Pathology.—No characteristic lesions are observed after death. The poison excites an inflammation of the respiratory mucous membrane, and probably irritates the peripheral filaments of the pneumogastric nerve, and so causes the paroxysmal cough. In fatal cases, pulmonary complications are usually discovered, such as catarrhal pneumonia, pulmonary collapse, and emphysema.

Symptoms.—There are three stages: (1) The catarrhal stage; (2) the paroxysmal stage; and (3) the stage of decline.

Catarrhal Stage.—The disease begins with the symptoms of coryza, and bronchial catarrh—slight fever, sneezing, running

from the nose, dry cough, and râles. But it does not respond to the ordinary remedies for catarrh, and after lasting one or two weeks passes into the paroxysmal stage.

Paroxysmal Stage.—The cough becomes more violent and paroxysmal. During the paroxysm the face is cyanosed, the eyes are injected, and the veins distended. The cough frequently induces vomiting, and, in severe cases, epistaxis or other hemorrhages. The close of the paroxysm is marked by a long-drawn, shrill, whooping inspiration due to the spasmodic closure of the glottis.

The number of paroxysms, or "kinks," varies from ten or twelve to forty or fifty in the twenty-four hours. From the forcible propulsion of the tongue against the lower incisors, an ulcer is frequently formed on the frænum. The duration of this stage is three or four weeks.

Stage of Decline.—The paroxysms grow less frequent and less violent and finally cease. Protracted cases are followed by anæmia and prostration.

Duration.—The entire duration of the disease is from a few weeks to four months.

Complications and Sequelæ.—Catarrhal pneumonia, pulmonary collapse, emphysema, hemorrhage into the conjunctiva, ear, or brain, and convulsions. Grave cases are sometimes followed by nephritis, chronic bronchitis, tuberculosis, or cancrum oris.

Diagnosis.—This can rarely be made with certainty during the catarrhal stage. Later, the paroxysmal cough ending in vomiting or in whooping is absolutely diagnostic.

Prognosis.—Guardedly favorable. Severe cases in the young and debilitated not infrequently prove fatal.

Treatment.—The child should be clad in flannel underclothes and carefully protected from changes of temperature. During the catarrhal or febrile stage the patient should be confined to bed. The diet should be light and nutritious. Counter-irritants, like iodine, applied to the chest seem useful. Quinine is a reliable tonic and may be employed throughout the disease. The ordinary expectorant mixtures are valueless. Local applications to the respiratory mucous membrane give much relief. One of the following remedies may be inhaled:

Creosote and chloroform, dilute peroxide of hydrogen, or a solution of menthol.

℞ Menthol, gr. xx;
Petrolat. liquid., f℥j.—M.
Sig.—Spray the naso-pharynx and inhale several times a day.

In very young children a solution of menthol may be inhaled from a cloth held under the chin. When paroxysms are violent the inhalation of a few drops of nitrite of amyl is desirable.

The following antispasmodic remedies appear to lessen the severity and the frequency of the paroxysms: belladonna, antipyrin, asafœtida, and bromoform (gtt. i–iv), potassium bromide.

℞ Sodii bromidi, ʒiss;
Tinct. belladonnæ, f℥j;
Glycerini, f℥ss;
Aquæ, q. s. ad f℥ij.—M.
Sig.—A teaspoonful every three or four hours.

Or—

℞ Antipyrin, gr. xl–lx;
Syr. tolutan., f℥j;
Aquæ q. s. ad f℥ij.—M.
Sig.—A teaspoonful every two or three hours.

INFLUENZA.

(La Grippe, Catarrhal Fever, Epidemic Catarrh.)

Definition.—An acute infectious disease, characterized by fever, extreme prostration, pain in the head and back, and generally by catarrh of the respiratory or gastro-intestinal tract.

Etiology.—The disease occurs in epidemics which usually have their origin in Russia, whence they spread with wonderful rapidity over both continents. The exciting cause is without doubt a small bacillus found in the sputum, and first discovered by Pfeiffer in 1892. When prevalent, no age and neither sex is exempt. One attack does not confer immunity from others.

Pathology.—Influenza does not often kill save by its complications. The latter are most frequently associated with

the respiratory tract, and consist of capillary bronchitis, catarrhal pneumonia, and croupous pneumonia.

Symptoms.—The disease begins abruptly with lassitude, malaise, chilliness, severe pain in the head and back, fever ranging between 101° and 103°, and extreme prostration, which is out of proportion to the fever and any existing local inflammation. The catarrhal symptoms are injection of the eyes, sneezing, hoarseness, and hard paroxysmal cough. In simple cases the temperature falls in two or three days by crisis, but complications not infrequently prolong the case for several weeks.

In some cases the catarrh of the respiratory tract is the chief feature; in others the gastro-intestinal tract is attacked, and the symptoms resemble cholera morbus; in a third group neuralgic pains in the head, back, and limbs are the most striking phenomena.

Complications.—Catarrhal pneumonia, croupous pneumonia, pleurisy, nephritis, neuritis, meningitis, and insanity.

Diagnosis. *Acute Bronchitis.*—The fever is not so high; there is little or no prostration; and the pains in the head and back are not nearly so marked as in influenza.

Typhoid Fever.—The gradual onset, typical temperature curve, epistaxis, diarrhœa, and rash will indicate typhoid fever.

Prognosis.—Uncomplicated cases nearly always recover. In the very old, and in those debilitated by chronic disease, influenza not infrequently proves fatal.

Treatment.—Absolute rest in bed and a liquid diet. As there is no specific, the treatment is symptomatic. Quinine is a useful stimulant, and when the stomach is irritable it may be given by the rectum.

The Pains. — Hot-water bags to the head and spine; morphine, or combinations of antipyrin or phenacetin with salol or salicin, thus:—

℞ Salol, ʒss;
Phenacetin, ʒj.—M.
Ft. in chart. No. xii.
Sig.—One every two hours.

Or—

℞ Quininæ salicylat., gr. xl;
Phenacetin, ʒi.—M.

In 20 capsules.
Sig.—One every two hours.

Or—

℞ Salicini, āā ʒij;
Phenacetin, ʒiss;
Olei gaulther., gtt. v;
Syr. acaciæ, fʒiij.—M.

Sig.—Teaspoonful every hour or two.

Heart-failure should be combated by alcohol and strychnine. Bronchial catarrh will require the remedies indicated in simple bronchitis. Sleep may be induced by opium, sulphonal, or bromide of potassium.

MUMPS.

(Epidemic Parotitis.)

Definition.—An acute contagious disease, characterized by inflammation of the parotid and other salivary glands.

Etiology.—The disease occurs sporadically and epidemically. It is most frequently observed in young children, but unprotected adults are not exempt. Males are more susceptible than females. The disease is highly contagious, and the virus is probably contained in the saliva, but it has not been isolated. One attack confers immunity from others.

Pathology.—As the disease is so seldom fatal very little opportunity is afforded for studying its intimate pathology. The parotid glands are the seat of an inflammatory infiltration, but suppuration does not occur. The inflammation shows a marked tendency to leave the parotids and to involve the testes in the male, or more rarely the mammæ or ovaries in the female.

Period of Incubation.—One to two weeks. 2–3 wks Osler

Symptoms.—The disease is ushered in with chilliness, malaise, and moderate fever (101°–104°), followed by swelling of one parotid gland. The swelling is observed below and in front of the ear, is pyriform in shape, and has a doughy feel. The surrounding tissues are œdematous, the submaxillary glands are likewise swollen, and the features may be dis-

torted beyond recognition. The movements of the jaw are restricted and painful. The saliva may be increased or diminished. In many cases the other parotid becomes similarly affected.

Often in the course of the disease the inflammation suddenly subsides in the parotid gland and reappears in the testicle in the male, or in the ovary or mamma in the female.

The duration of the disease is usually five or six days.

Complications.—Hyperpyrexia, metastasis to the testicle or ovary, and meningitis. Atrophy of the testicle rarely follows.

Prognosis.—Favorable.

Treatment.—Rest in bed. Mild febrifuges may be given internally. Locally, lead-water and laudanum, or some rubefacient liniment like the following, may be employed :—

℞ Tinct. iodi,
Tinct. aconit. rad.,
Tinct. opii, āā f℥ij ;
Liniment. chloroform., q. s. ad f℥iij.—M.
Sig.—Apply externally and cover with cotton-wool.

The swollen testicle should be elevated and covered with lint saturated with lead-water and laudanum. If the swelling persists, an ointment of mercury, belladonna, and ichthyol will be found useful.

CHOLERA.

(Asiatic Cholera, Epidemic Cholera, Malignant Cholera.)

Definition.—An acute infectious disease, generally epidemic, excited by Koch's comma-bacillus, and characterized by vomiting and purging of a serous material, painful cramps, and collapse.

Etiology.—Cholera has its origin in India, and is carried thence to other parts of the world. The exciting cause is the comma-bacillus of Koch ; this usually has the form of a slightly-curved rod, but it is occasionally S-shaped. The rice-water evacuations only contain the bacilli, which, under favorable conditions, continue to grow outside of the body, and by gaining entrance into the healthy system propagate the

disease. The disease always spreads along the lines of traffic, hence epidemics nearly always begin at the sea-coast and extend inland. Cholera is slightly, if at all, contagious; like typhoid fever, the poison is not carried through air, but chiefly through drinking-water. Laundresses and nurses, from their contact with the evacuations, readily acquire the disease. Epidemics are more frequent in summer than in winter. No age is exempt, but the old are more susceptible than the young. The intemperate, the debilitated, and those suffering with gastro-intestinal catarrh are especially predisposed.

Pathology.—The body is shrivelled; movements of the corpse are sometimes observed; rigor mortis is marked and prolonged. The tissues are dry, and the large veins and right side of the heart contain thick, dark blood. The serous cavities are empty and their surfaces sticky. The intestines contain more or less rice-water fluid, from which cultures of bacilli can be made.

The mucous membrane has a pinkish color and is often the seat of ecchymoses; the solitary and Peyer's glands are swollen. Frequently extensive desquamation of the epithelial lining is observed, but this is usually regarded as a post-mortem change. The kidneys reveal evidences of parenchymatous inflammation; the liver is the seat of fatty degeneration.

As the lesions are not sufficient to explain the clinical phenomena, it has been suggested by Koch that the bacilli create a poison the absorption of which causes the grave symptoms.

Period of Incubation.—A few hours to several days.

Symptoms.—The severity of the symptoms varies considerably. In well-marked, but favorable, cases there are three stages: (1) Invasion; (2) algid or collapse; (3) reaction.

Stage of Invasion.—The disease usually begins with malaise, headache, diarrhœa, rumbling noises in the intestines, and colic. Frequently these symptoms continue a few days and then subside; such cases are termed *cholerine*, and are as infectious as the fully-developed disease.

Stage of Collapse.—The diarrhœa grows more marked; the evacuations become copious, lose their feculent character, assume a rice-water appearance, and are discharged forcibly but without pain. Vomiting soon develops, and the ejected material

resembles that passed by the bowel. Thirst is unquenchable. Severe cramps seize the muscles of the calves of the legs, thighs, arms, and abdomen. The surface is cold and covered with a clammy sweat; the breath is cool; the temperature in the axilla ranges from 95° to 85°, while in the rectum it may rise to 103° or more. The voice is husky and finally reduced to a whisper; the respirations are quickened; the pulse becomes more and more feeble; the body is livid and shrivelled; the hands resemble those of a washerwoman; the features are pinched and sometimes distorted; the eyes are frightfully sunken. The urine is more or less suppressed, and the little that is passed generally contains albumin and a trace of sugar. Consciousness is usually retained until near the end, when coma sets in.

The duration of this stage is from a few hours to two days.

Stage of Reaction.—Sometimes, even when death seems imminent, the surface-temperature begins to rise; the urine increases; the pulse strengthens; the vomiting ceases; the evacuations from the bowels become less frequent and begin to assume a feculent character, and convalescence is established.

Occasionally, instead of convalescence, symptoms of a typhoid type develop, such as moderate fever, a brown, fissured tongue, subsultus, muttering delirium, and coma. This condition, which is generally fatal, has been regarded as uræmic.

Cholera Sicca.—In very violent cases collapse and death may follow without there having been any evacuation. After death the intestines contain rice-water fluid, which was not discharged during life probably on account of paralysis of the muscular coat of the bowel.

Complications and Sequelæ.—Nephritis, pneumonia, pleurisy, parotitis, ulceration of the cornea, diphtheritic inflammation of the throat and fauces, abscesses, and local gangrene.

Diagnosis. *Cholera Morbus.*—This is always sporadic; the discharges are bilious in character; a history of dietetic errors and of exposure can usually be obtained; and the comma-bacilli are not detected in the discharges.

Prognosis.—Generally unfavorable. The mortality averages about 50 per cent. In the old, young, debilitated, and

intemperate it is very fatal. In individual cases, early collapse and a low surface temperature are unfavorable conditions.

TREATMENT. *Prevention.*—This includes the isolation of the sick; absolute cleanliness; the disinfection of excreta and soiled bed-clothes; the thorough boiling of all water that is to be used for drinking purposes; the use of a bland, unirritating diet; the avoidance of overwork, exposure, and undue excitement; and the prompt treatment of any gastro-intestinal disturbance that may arise.

The Attack.—The violent vomiting and purging and the cramps call for morphine; this is best administered hypodermically. There are no specifics. A remedy frequently recommended by competent observers is sulphuric acid, which may be given with laudanum or chlorodyne. Thirst is best assuaged by cracked ice *ad libitum* and acidulated drinks. For the vomiting a mustard poultice may be applied to the epigastrium, and iced champagne, carbolic acid, creosote, or dilute hydrocyanic acid may be given internally. For the cramps the application of hot-water bags, warm fomentations, or the rubbing in of warm oil may be useful; when they are very severe a few whiffs of chloroform may be employed. When the pulse weakens, stimulants like alcohol, ether, and ammonia should be given freely.

Copious warm-water enemata containing tannic acid (1 per cent.) and laudanum are highly recommended for the purging.

The low temperature must be combated by the use of hot blankets, or, better still, by immersion in warm baths (98° to 104°). In collapse, subcutaneous or intravenous injections of saline solutions have been highly recommended. The following solution, which is well spoken of by Fagge, may be injected directly into the veins, or may be allowed to flow through a rubber tube attached to an aspirating canula, and to enter the subcutaneous tissue by its own pressure:—

℞ Sodii phos., gr. iij;
Sodii chlorid., ʒj;
Potass. chlorid., gr. vj;
Sodii carb., gr. xx;
Alcohol, fʒij;
Aquæ destil., fʒxx.—M.

The fluid should be warm, and the injection should be continued until the pulse strengthens; as much as eighty ounces may be introduced at one time.

The diet should consist of the following: Light broths, milk with carbonated water, koumiss, wine-whey, thin gruels, and frozen blocks of beef-tea.

TETANUS.

(Lockjaw.)

Definition.—An acute infectious disease excited by a special bacillus, and characterized by painful tonic spasms of the voluntary muscles.

Etiology.—In the tropics, especially in the colored race, the disease often arises idiopathically. In temperate climates the poison nearly always gains entrance through a wound. Lacerated and punctured wounds, frost-bites, and burns are especially liable to become infected. Exposure to cold and wet after traumatism seems to predispose. Since the introduction of antiseptic surgery tetanus is less common than formerly.

The exciting cause is a special microörganism—the *tetanus bacillus.*

Pathology.—Congestion of the spinal cord and of the nerves leading to the wound.

Symptoms.—The disease begins with a feeling of rigidity in the muscles of the neck and lower jaw; by degrees the muscles of the back, abdomen, and lower extremities are similarly involved. The brow is wrinkled, the corners of the mouth are drawn upwards (*risus sardonicus*), the jaws are tightly closed (*trismus*), and the body becomes arched, the patient resting on his head and heels (*opisthotonos*). There is extreme hyperæsthesia, so that the slightest touch causes a violent exacerbation of the spasm, which is attended by excruciating pain. If the respiratory muscles are involved, there is intense dyspnœa. The temperature usually remains normal until just before death, when it may rise to 107° or more. The mind is clear to the end. The duration is from a few days to several weeks.

DIAGNOSIS. *Strychnia-poisoning.*—The history of the case, the complete relaxation between the spasms, and the late involvement of the jaw will indicate strychnia-poisoning.

Tetany.—The history, the paroxysmal character of the spasms, the involvement of the hands, and the *escape* of the trunk and jaw will serve to distinguish tetany from tetanus.

PROGNOSIS.—Unfavorable. Slight involvement of the muscles of the trunk, absence of fever, and a slow course are favorable features.

TREATMENT.—The wound should be rendered aseptic. Morphine is indicated for the relief of the pain. Bromide of potassium (ʒj every two hours) and chloral should be used to control the convulsions. When asphyxia is threatened by the violence of the spasm, inhalations of chloroform should be employed. When the patient is unable to swallow, he must be fed through the nose or by the rectum.

Antitoxins derived from the blood of animals which have been rendered immune will doubtless prove to be a valuable addition to the therapy of this dread disease.

DENGUE.

(Break-bone Fever, Dandy Fever.)

DEFINITION.—An acute infectious disease, characterized by pains in the muscles and joints, a variable rash, and a febrile course of two paroxysms.

ETIOLOGY.—Dengue is confined almost entirely to hot climates. Although it occurs in epidemics, its contagiousness is still a matter of dispute.

PERIOD OF INCUBATION.—Three to five days.

SYMPTOMS.—The invasion is usually sudden and is attended with lassitude, chilliness, headache, intense pain in the muscles and joints, and high fever. The latter rises rapidly and often reaches a maximum of 104°–105° in a few hours. The pulse is rapid and full; the respirations are accelerated; the mind is often delirious; the urine is scanty; the joints are swollen and stiff. In two or three days the temperature falls, and an afebrile period follows in which the patient is free from pain, but is profoundly prostrated. During the remission a roseo-

lar or a diffuse erythematous rash generally appears; this lasts two or three days and is followed by slight desquamation. Shortly after the subsidence of the rash, the fever and pains again return, and persist for two or three days when convalescence begins.

DIAGNOSIS.—*Acute Rheumatism.* The prevalence of an epidemic, and the distinct remission will usually render the diagnosis apparent.

PROGNOSIS.—Favorable.

TREATMENT.—There is no specific remedy. High fever should be controlled by the external application of cold or by the use of antipyrin. Morphine, salol, antipyrin, or phenacetin may be employed to relieve pain. Prostration must be combated by stimulants, like alcohol, quinine, and strychnine.

HYDROPHOBIA.

(Rabies.)

DEFINITION.—A disease of dogs and kindred animals, communicated to man by direct inoculation, and characterized by slight fever, painful spasm of the muscles of the throat, delirium, paralysis, and coma.

ETIOLOGY.—Rabies invariably results from the bite of a rabid animal, generally a dog. In the animal the disease is characterized by depression of spirits, loss of appetite, followed by excitement, aimless roving, a morbid desire to bite, and finally by paralysis and death from exhaustion. The poison is contained in the saliva and blood. Pasteur has induced the disease by direct inoculation, and has found that the virus is attenuated by passing several times through the monkey. Bites on the face and on exposed parts are particularly liable to be followed by infection.

PATHOLOGY.—Intense congestion of the spinal cord and of the respiratory mucous membrane.

PERIOD OF INCUBATION.—Six weeks to six months.

SYMPTOMS. *First Stage.*—Depression of spirits, restlessness, slight difficulty in swallowing, and pain in the wound or cicatrix. In a few days the stage of excitement begins.

Second Stage.—Clonic convulsions, involving especially the muscles of the throat, occurring spontaneously or excited by drinking or by the sight of water; hyperæsthesia, delirium, moderate fever, and salivation. This stage lasts a few days, and is followed by paralysis.

Third Stage.—The pulse weakens; the convulsions cease; the patient lies motionless; the mind becomes clouded; and death results in twelve or twenty-four hours from exhaustion.

Diagnosis.—*Hysteria* in persons who have been bitten may simulate hydrophobia. Such persons often bark, try to bite, and manifest other symptoms which are not noted in hydrophobia.

Prognosis.—Invariably fatal.

Treatment. *Prophylaxis.*—Suspicious bites should be thoroughly disinfected and cauterized by the hot iron or caustic potash, after which the patient should be sent to an institute where inoculation may be practised after the method of Pasteur.

The Attack.—Palliative. For the convulsive seizures morphine may be employed hypodermically, and chloroform by inhalation. The strength may be sustained by rectal alimentation.

CONSTITUTIONAL DISEASES.

RHEUMATIC FEVER.

(Acute Articular Rheumatism, Inflammatory Rheumatism.)

DEFINITION.—An acute general disease, characterized by irregular fever, acid sweats, inflammation of the joints, and a marked tendency to involve the heart.

ETIOLOGY.—Heredity, temperate zone, occupations which necessitate exposure to cold and wet, early life (15–40), and one attack are predisposing factors. The disease is usually precipitated by sudden chilling of the body.

The exciting cause is still unknown. Some regard it as a neurosis; others believe it to be infectious, and classify it with pneumonia, erysipelas, and similar diseases; while still others attribute it to deranged metabolism. According to the last theory, the nitrogenous products, instead of being converted into urea, are transformed into lactic acid, uric acid, and other allied substances, and these deleterious agents are responsible for the symptoms.

PATHOLOGY.—The ligaments and the synovial membrane and its fringes are congested and swollen. The synovial sac is filled with a turbid fluid. The cartilages are roughened and occasionally ulcerated. Generally the process ends in resolution; sometimes the surrounding tissues become infiltrated with inflammatory lymph, and false anchylosis results; rarely, suppuration of the joint follows. Sometimes small, subcutaneous, fibrous nodules are found near the joints and large tendons. The blood shows an excess of fibrin and a considerable diminution of the red corpuscles. Fibrinous clots are often found in the heart and great bloodvessels.

Secondary inflammations are frequently discovered, such as endocarditis, pericarditis, pleurisy, or pneumonia.

Symptoms.—The symptoms vary much in their severity. The disease usually begins abruptly, or more rarely follows such prodromes as malaise, chilliness, and sore throat. The large joints, especially the symmetrical ones, are usually affected; they are slightly reddened, swollen, exquisitely painful, and tender to the touch. The inflammation shows a marked tendency not only to spread from joint to joint, but to disappear abruptly in one while it attacks another. The joints most commonly involved are the knees, elbows, ankles, and wrist; but no joint is exempt. In severe cases the muscles are painful, tender, and sometimes rigid. The fever rises rapidly to a moderate height (102°–103°), and is indefinite in its duration and extremely irregular in its course. Perspiration is often copious, has a peculiar sour smell and an acid reaction. The urine is scanty, high-colored, and on standing throws down an abundant sediment of urates and uric acid. The tongue is heavily coated; the appetite is lost; and the bowels are constipated. The face is at first flushed, but as the disease advances it becomes decidedly pale from anæmia.

The duration is indefinite, varying from a few days to several weeks.

Complications.—Endocarditis (in 40 per cent. of all cases); pleurisy; pericarditis; pneumonia; hyperpyrexia (106°–109°), which is often associated with maniacal delirium; chorea; iritis; meningitis; and certain cutaneous phenomena, such as urticaria, purpura, erythema nodosum, and subcutaneous fibrous nodules.

Diagnosis. *Septic Arthritis.*—This may be recognized by its association with some other septic process and by the special tendency of the inflammation to end in suppuration, which is a very rare termination of rheumatic fever.

Gonorrhœal Rheumatism.—This may be recognized by the history, by its obstinate character, and by its tendency to involve, not only large joints, but certain small joints which are rarely affected in rheumatic fever, like the sterno-clavicular, temporo-maxillary, and sacro-iliac.

Rheumatoid Arthritis.—This begins in the small joints, attacks one after another, leads to permanent deformity, is not associated with fever and sweats, and shows no tendency to involve the heart.

Gout.—This occurs later in life, usually involves the great toe, and lacks high fever, acid sweats, and the tendency to heart complications.

Prognosis.—Guarded. Most cases end in recovery; some in chronic rheumatism; a very small number die of exhaustion, or some complication, such as hyperpyrexia. It is very prone to relapse and to recur. The most frequent complication is endocarditis; this may never give rise to trouble, but frequently it leads to slow thickening or retraction of the valves and to all the phenomena of chronic heart disease.

Treatment.—Absolute rest in a room well-ventilated but free from draft; the patient should lie between blankets. The diet should consist mainly of milk and light broths; meat should be interdicted. The free use of lemonade or mineral waters should be encouraged. Opium, phenacetin, or antipyrin may be required to relieve the pain.

Two remedies have considerable power in controlling the disease: salicyl compounds, and alkalies, like the salts of potassium; these remedies may be given separately or in combination. The salicylates relieve the pain, but do not prevent relapses or cardiac complications; the alkalies apparently lessen the tendency to endocarditis.

Salicylic acid (gr. x in capsules) or salicylate of sodium (gr. x–xx) may be given every two hours. Large doses may excite nausea and ringing in the ears.

℞ Sodii salicylat., ʒij;
Tinct. cardamom. comp., fʒiv;
Glycerin., fʒij;
Aquæ q. s. ad fʒiv.—M.
Sig.—A tablespoonful every two hours.

The oil of gaultheria (♏x every two hours) is another salicyl compound of decided value. If alkalies are employed, half a drachm of bicarbonate of potassium may be administered every two hours until the urine becomes distinctly alkaline. It is a good plan to combine alkalies with salicylates, thus:—

℞ Sodii salicylat., ʒij ;
Potass. bicarb., ʒiij ;
Glycerini,
Tinct. cardamom. comp., āā fʒss ;
Aquæ q. s. ad fʒv.—M.
Sig.—A tablespoonful every two hours.

When there is much anæmia Basham's mixture (ʒj–ʒss) may be given with the salicylate, or the following combination may be employed :—

℞ Acid. salicylic., ʒss ;
Ferri pyrophosphat., ʒj ;
Sodii phosphatis, ʒx ;
Aquæ, fʒvj.—M. (Peabody.)
Sig.—Tablespoonful every two hours until relieved.

Local Treatment.—The joints may be painted with iodine and wrapped in cotton-wool. In severe cases small blisters are of great utility. Chloroform liniment, aconite liniment, lead-water and laudanum are also efficient remedies. The salicyl preparations, when applied locally, often relieve the pain better than any other remedy. The following mixture may be employed :—

℞ Æther.,
Alcohol.,
Ol. gaultheriæ, āā ʒj ;
Lin. saponis q. s. ad Oj.—M.
Sig.—Apply locally.
Or—

℞ Ol. gaultheriæ,
Ol. olivæ,
Lin. saponis,
Tinct. aconit.,
Tinct. opii, āā fʒiss.—M.
Ft. liniment.
Sig.—Apply locally.

Sometimes ichthyol proves serviceable.

℞ Ichthyol, ʒij ;
Ext. belladonnæ, ʒj ;
Vaselin., ʒij.—M.
Sig.—Apply locally.

Hyperpyrexia. — This should be treated promptly by the cold pack or the cold bath.

Endocarditis.—This usually causes no subjective disturbance and the general treatment need not be modified. When the pulse is rapid and irregular, and the patient complains of præcordial distress, a blister may be applied and digitalis may be given internally. Absorbents like the iodide of potassium are useless. Convalescence should be protracted so as to allow time for perfect compensation.

Convalescence.—Such tonics as iron, quinine, and strychnine are useful during this period.

CHRONIC ARTICULAR RHEUMATISM.

Etiology.—It usually begins as a chronic affection. Heredity, advanced years, and habitual exposure to cold and wet are the predisposing factors. It rarely results from an acute attack.

Pathology.—The fibrous structures around the joint are greatly thickened, so that in long-standing cases the movements are restricted; the neighboring muscles are wasted from disuse; and the nerves often reveal evidences of neuritis.

Symptoms.—Pain, stiffness, deformity, and creaking of the joints are the usual phenomena. Several joints are commonly affected, and the disease shows no predilection for any particular joint. The symptoms grow worse on the approach of stormy weather, and at such times exacerbations are liable to occur, in which the joints become swollen and tender. The duration is indefinite.

Complications.—Arterial degeneration and chronic endocarditis.

Prognosis.—Generally unfavorable. Much relief may follow persistent and judicious treatment, but perfect cure is rarely attainable.

Treatment.—Especial attention should be given to the hygiene, particularly as regards diet, bathing, clothing, exercise, and occupation. A change of residence to a dry, warm, and equable climate may effect a cure. The tone of the system is often reduced; hence, tonics like iron, quinine, strychnine, and arsenic may be of considerable value. The special remedies are iodide of potassium, guaiac, sulphur, salicylic acid,

and alkalies like the salts of potassium and lithium. Mineral waters are sometimes useful.

℞ Liq. potass. arsenitis, f℥ij ;
Potass. iodid., ℥ij ;
Syr. simp., f℥iij.—M. (Da Costa.)
Sig.—A teaspoonful three times a day in water after meals.

OTHER MANIFESTATIONS OF RHEUMATISM.

Muscular Rheumatism (*myalgia, myodynia*).—An affection of the voluntary muscles, characterized by pain, tenderness, and rigidity.

Types.—Different names have been applied according to the location, namely: *Torticollis*, or *wry-neck*, when it involves the sterno-cleido-mastoid muscles; *lumbago*, when it involves the lumbar muscles; *pleurodynia*, when it involves the intercostals; and *cephalodynia*, when it involves the occipito-frontalis.

Etiology—The gouty or rheumatic diathesis is a predisposing cause. Exposure to cold and wet or muscular strain usually excites it.

Symptoms.—Pain is the chief symptom; it is made worse by use of the muscles, and is associated with tenderness which is especially marked at the tendinous origins and insertions of the muscles. Sometimes the muscles are contracted and rigid; this is particularly the case in torticollis, or wry-neck.

Torticollis.—The head is fixed and inclined to one side; every effort to turn it is attended with sharp pain.

Lumbago.—There is a dull, aching pain across the loins. Turning the body or rising from the sitting posture causes an exacerbation, which is sometimes so severe that the patient cries out. Care must be taken to distinguish it from renal calculus, Pott's disease, aneurism, perinephritis, and uterine or ovarian disease.

Pleurodynia.—The pain is felt in the side, and is increased by deep breathing, coughing, or twisting the body; the respirations are restricted on the affected side. There is diffuse tenderness to the touch. The absence of fever and of physical signs will serve to distinguish it from *pleurisy*.

The absence of tender spots where the nerves make their exit from the muscular coverings, the fact that the pain does not follow closely the distribution of the nerves, and that the pain is increased by movement, will serve to distinguish pleurodynia from *intercostal neuralgia*.

Cephalodynia.—This is characterized by a superficial head pain which is increased by moving the scalp and which is associated with tenderness on pressure.

PROGNOSIS.—Favorable under judicious and persistent treatment.

TREATMENT.—The affected muscles should be put at rest. In pleurodynia this is best accomplished by strapping the affected side as for fracture of the ribs. In lumbago a large piece of adhesive plaster may be applied from the floating ribs to the iliac crests. In mild cases the thorough application of liniments containing chloroform, aconite, belladonna, and laudanum will be all that is required. In other cases prompt relief often follows the injection of morphine (gr. $\frac{1}{8}$) with atropine (gr. $\frac{1}{125}$), directly into the muscle. The continued current is sometimes useful. The introduction of needles, three or four inches long, deeply into the muscles (acupuncture) occasionally gives brilliant results.

Internally, in acute cases, chloride of ammonium (gr. x four times daily) may prove efficient. In chronic cases, iodide of potassium, guaiac, colchicum, and the salts of lithium are the remedies usually employed. Gelsemium pushed to its physiological limit has been successful when other remedies have failed.

Neural Manifestation.—Rheumatism appears to be a frequent cause of neuritis.

Rheumatic Affections of Mucous Membranes.—It must be borne in mind that pharyngitis, tonsillitis, laryngitis, and bronchitis are sometimes dependent upon a rheumatic diathesis.

Rheumatic Affections of Serous Membranes.—Endocarditis, pericarditis, pleuritis, iritis, and peritonitis may be excited by rheumatism.

Cutaneous Manifestations.—Purpura, urticaria, and erythema nodosum are sometimes associated with rheumatism.

GOUT.

(Podagra.)

DEFINITION.—A general disease, characterized by varied constitutional disturbances, the presence of uric acid in the blood, the deposition of urate of soda in the fibrous structures of the joints, and recurrent attacks of acute arthritis.

ETIOLOGY.—Middle and advanced life, male sex, heredity, a rich diet and the indulgence in liquors (especially malt liquors and strong wines), want of exercise, and working in lead are general predisposing factors.

PATHOLOGY.—The blood contains uric acid, and the fibrous structures of the joint are the seat of a deposit of urate of soda. It is probable that from defective nerve-power the tissues generally are unable to perfect the metabolism of nitrogenous products into urea, and that uric acid and allied substances are thus formed. According to Ebstein, the uric acid excites a necrosis of the cartilages, whereupon the urates are crystallized out and deposited.

The cartilages lose their pearly appearance and become lustreless and infiltrated with salts; similar opacities appear in the synovial membrane; later rounded masses of urate of soda (tophi), varying in size from a pea to a marble, accumulate in the tissues surrounding the joint and may ulcerate through the skin and be discharged. The fibrous structures become brittle and undergo destructive changes. The joint becomes irregularly enlarged, stiff, and finally anchylosed. The metatarso-phalangeal joint of the great toe, especially the right one, is first affected, but soon other small joints are involved. Gouty deposits are sometimes found along the tendons, beneath the peritoneum, in the perichondrium of the ear, and in the tarsal cartilages.

The kidneys are generally the seat of a chronic interstitial inflammation, and section frequently reveals a deposit of urates at the apices of the pyramids (gouty kidney). The arteries are sclerosed and the left side of the heart is hypertrophied.

SYMPTOMS. **Acute Gout.**—Such prodromes as restlessness, insomnia, moroseness, and irritability of temper may precede the

attack. The arthritic phenomena usually appear suddenly in the early morning hours and are characterized by pain and swelling in the ball of the great toe. The affected joint is exquisitely painful and tender, so that the slightest pressure cannot be borne; it is of a reddish-purple color; its surface is glazed; and the neighboring veins are full and distinct.

The constitutional symptoms are restlessness, chilliness, moderate fever, perspiration, constipation, and scanty high-colored urine, which contains, *during the paroxysm*, less urates than in health. Towards daylight the symptoms abate and the patient falls to sleep. During the day he is comparatively comfortable, but there are severe exacerbations for several successive nights. At first the attacks may be a year apart, but as they multiply the interval grows less, until finally the patient is seldom entirely free from suffering.

Retrocedent Gout.—This term is applied to a condition in which the arthritic phenomena suddenly subside and grave gastric, cardiac, or cerebral symptoms follow.

Chronic Gout.—The joints are affected one by one, and become stiff, irregularly enlarged, and deformed. Chalk-stones, or tophi, sometimes ulcerate their way through the skin and are discharged. Similar deposits are frequently found along the tendons and in the helix of the ear. The constitutional symptoms vary much in severity and in character.

Nervous Phenomena.—Vertigo, headache, insomnia, irritability of temper, and hypochondriasis.

Gastro-intestinal Phenomena.—Perverted appetite, dyspepsia, constipation, and a tendency to hemorrhoids.

Urinary Phenomena.—The urine is at first scanty, high-colored, and throws down an abundant brick-dust sediment; but ultimately interstitial nephritis develops and the urine becomes pale, copious, of a low specific gravity, and contains albumin and hyaline casts. Glycosuria is also frequently observed.

Circulatory Phenomena.—High arterial tension, accentuation of the aortic second sound, and later, arterio-sclerosis and hypertrophy of the left ventricle.

Complications and Sequelæ.—Interstitial nephritis, arterio-sclerosis, hypertrophy of the heart, apoplexy, chronic bronchitis, and cutaneous eruptions, especially eczema.

Diagnosis.—The symptoms of acute gout are so characteristic that an error in diagnosis is scarcely possible.

Chronic gout may be mistaken for *chronic rheumatism;* but the former attacks especially the small joints; it begins in the great toe; the blood contains an excess of uric acid; and the symptoms are not so much influenced by atmospheric changes as by diet.

Prognosis.—As regards the acute form, the prognosis is good. The liability to arterial degeneration and to nephritis, and the difficulty in securing coöperation in carrying out the treatment render the prognosis of chronic gout rather unfavorable.

Treatment. *The Acute Attack.*—The best remedy is colchicum; ten to twenty drops of the wine well diluted should be given every two hours, and stopped as soon as the symptoms subside. Alkalies are valuable adjuncts, and the salts of potassium or of lithium may be given with the colchicum. Quinine is also useful; it may be given in doses of five grains thrice daily. The free use of water should be encouraged, and a water containing lithium, like the Buffalo lithia water, may be recommended. Constipation should be relieved by a full dose of blue mass or a saline draught. Opium may be required for the relief of the pain. The affected part should be elevated and wrapped in cotton-wool, or covered with warm fomentations or with cloths soaked in lead-water and laudanum. The diet should be light and non-stimulating.

Chronic Gout.—The diet must be restricted and carefully arranged for each patient. Light meats, fish, eggs, and oysters may be used in moderation; sweet fruits should be avoided; starches and sugars must be limited; and the use of liquors interdicted. The condition of the tongue, stomach, and urine will indicate the value of this or that dietary. Mineral waters are often serviceable, and Carlsbad, Vichy, and Buffalo lithia are among the best. Their utility will be enhanced by the addition of a teaspoonful of some effervescing salt of lithium to each potation. A free secretion of the skin should be encour-

aged by frequent bathing followed by friction. The bowels should be kept regular by salines or by the occasional use of a mercurial laxative. Graduated exercise holds a prominent place in the therapy of gout. When the digestive powers are particularly weak, mineral acids with strychnine will prove useful. General tonics are sometimes indicated. The special remedies are colchicum, lithium, and iodide of potassium.

℞ Vini sem. colchici, f℥ss;
Potass. iodidi, ʒij;
Liq. potass., f℥iss;
Tr. zingiberis, f℥ij.—M. (Hodgson.)
Sig.—A teaspoonful twice daily in warm water.

Or small doses of colchicum may be given with—

℞ Lithii benzoat., ℈ij;
Aq. cinnamom., f℥ijss.—M. (Jaccoud.)
Sig.—A teaspoonful in a wineglass of water every four hours.

The arthritic condition is best treated by careful massage and warm sulphur baths.

RHEUMATOID ARTHRITIS.

(Arthritis Deformans, Rheumatic Gout.)

Definition.—A chronic affection of the joints characterized by destruction of the cartilages, new osseous formations, immobility, and deformity.

Etiology.—Heredity; early adult life; female sex; continued emotional disturbances, as anxiety and grief; enfeeblement of health from bad hygienic environment, prolonged lactation, and from frequent pregnancies, are the predisposing causes.

Pathology.—Many look upon rheumatoid arthritis as a disease which is related both to gout and rheumatism. Others regard it as a neurosis and allied to the arthropathies which are met with in chronic affections of the spinal cord.

The cells of the cartilages and of the synovial membrane proliferate and lead to villous or nodular outgrowths. The central portions of the cartilages ultimately wear away and leave the bones exposed. The heads of the bones become

smooth and hard like ivory, and thickened from exostoses. The synovial membrane and periarticular tissues are likewise thickened and sometimes infiltrated with bony products. The surrounding muscles are generally atrophied. All joints are liable to be affected.

Symptoms.—It may be either acute or chronic, the latter being the more common form. In the *acute form* several joints are simultaneously involved; they become swollen, painful, and slightly reddened. There is moderate fever. The symptoms soon subside, to reappear, however, at frequent intervals.

In the *chronic form,* the hands, particularly the metacarpophalangeal joints, are usually first affected; then the wrists, knees, toes, jaws, and spine. Symmetrical joints are usually attacked. The symptoms are: Swelling, pain, immobility, and deformity; the joints are stiff and creak when moved; later complete anchylosis develops; the muscles waste and contractures increase the deformity. In advanced cases the fingers are bent backward, often locked, and turned toward the ulnar side; the thighs are drawn up; the legs are adducted and flexed. The patient may be a helpless invalid for many years.

Diagnosis. *Gout.*—The circumstances under which gout develops; the history of an acute attack in the great toe; the presence of uric acid in the blood; the presence of urate of soda in the joints and in the cartilages of the ear will serve to distinguish the two diseases.

Chronic Rheumatism.—Unlike chronic rheumatism, rheumatoid arthritis begins in the small joints, passes from joint to joint, and leaves permanent deformity.

Prognosis.—Unfavorable. Sometimes the disease is local and remains in one joint (mono-articular form). Generally several joints are affected, and while judicious and persistent treatment may retard the progress of the disease, a cure is rarely attainable.

Treatment.—Good hygiene. Tonics like iron, arsenic, phosphorus, and cod-liver oil are useful. The most good is to be expected from local treatment, which consists of massage, electricity, steam baths, and inunctions of preparations containing iodine or mercury.

RICKETS.

(Rachitis.)

DEFINITION.—A constitutional disease of early childhood, characterized chiefly by defective nutrition of the osseous structures.

ETIOLOGY.—Rickets is rarely congenital; it usually develops between the first and second years. Poverty, artificial feeding, and bad hygienic conditions are the predisposing causes.

PATHOLOGY.—The most marked changes are observed in the long bones and ribs. The cartilaginous lamina between the epiphysis and the shaft are considerably thickened, and are spongy and irregular in outline; microscopic examination reveals an excessive proliferation of the cartilage-cells with scanty calcification. The periosteum is thickened and highly vascular, and when stripped off soft porous bone is exposed. The bones are soft, being extremely deficient in lime-salts; when ossification finally results the bones become heavy, large, and irregular in outline; these changes correspond to the clinical phenomena—bow-legs, knock-knees, spinal curvature, pigeon-breast, and square cranium.

The liver and spleen are often considerably enlarged.

SYMPTOMS.—The early symptoms are: Restlessness and slight fever at night; free perspiration about the head; diffuse soreness and tenderness of the body; pallor; slight diarrhœa; enlargement of the liver and spleen; delayed dentition and the eruption of badly-formed teeth.

Skeletal Phenomena.—The head is large and more or less square in outline; careful palpation may detect soft areas. The sides of the thorax are flattened; the sternum is prominent; nodules can be felt at the sternal ends of the ribs—"rachitic rosary"; there may be a distinct transverse groove at the level of the ensiform cartilage; the spinal column is frequently curved antero-posteriorly or laterally; the long bones are curved and prominent at their extremities.

COMPLICATIONS.—Green-stick fractures, convulsions, laryngismus stridulus, paresis of the extremities, and acute pulmo-

nary diseases. In women the rachitic pelvis may seriously complicate labor.

Prognosis.—Rachitis does not kill directly, but death is not uncommon from intercurrent disease. Under good hygienic conditions recovery, with more or less deformity, generally follows.

Treatment.—The general nutrition must be improved by placing the child under the best hygienic conditions. Eggs, pure milk, malt, and broths should be recommended. Cod-liver oil is a valuable nutrient tonic. Iron is indicated for the anæmia. The lack of calcareous material in the bones should be supplied by the administration of phosphorus and lime-salts.

℞ Syr. ferri iodid., f℥iss;
Mist. ol. morrhuæ et
Lactophos. calcis, q. s. ad f℥iij.—M. (Starr.)
Sig.—From one-half to a teaspoonful three times a day.

℞ Elixir. phosphori, f℥iiiss;
Ol. morrhuæ, f℥ij;
Pulv. acaciæ, q. s.;
Ol. sassafras, gtt. xv;
Aquæ q. s. ad f℥iv.—M.
Sig.—One to two teaspoonfuls three times a day.

LITHÆMIA.

(Lithic-acid Diathesis, Uric-acid Diathesis, Latent Gout.)

Definition.—A constitutional disease dependent upon malassimilation of nitrogenous products and the formation of uric acid and allied substances instead of urea, and characterized by an excess of uric acid in the urine, and varied digestive, circulatory, and nervous phenomena.

Etiology.—Gout with an acute arthritic expression is uncommon in America, but latent gout, or lithæmia, is extremely common. Impaired digestion, insufficient exercise, mental strain, and over-eating are the usual causes.

Symptoms. *Gastro-intestinal Phenomena.*—The tongue is generally coated and the breath heavy; the appetite is variable, sometimes it is lost, at others it is inordinate; acid eructations,

"heartburn," and flatulence are frequent gastric symptoms; the bowels are usually constipated.

Urinary Phenomena.—The urine is scanty, high-colored, of high specific gravity (1025 – 1035), and on standing throws down an abundant brick-dust sediment. The solids render the urine irritating, so that dull aching in the loins and burning in the penis after micturition are common symptoms. A trace of sugar is sometimes detected on chemical examination. The urine often stains the clothes red.

Circulatory Phenomena.—High arterial tension, accentuation of the aortic second sound, and a tendency to atheroma.

Nervous Phenomena.—Headache, vertigo, disturbed sleep, tinnitus aurium, depression of spirits, failure of memory, loss of energy, irritability, and neuralgic pain in various parts of the body.

Sequelæ. — Arterial degeneration, interstitial nephritis, hepatic cirrhosis, gastritis, renal or vesical calculi.

Diagnosis.—This rests on the general symptoms and the analysis of the urine.

Prognosis. — Favorable under prolonged and judicious treatment.

Treatment.—Special attention must be given to the diet. It is a mistake to cut off all nitrogenous foods, for often the chief difficulty is in digesting the starches and sugars. Light meats, green vegetables, eggs, and oysters are admissible. The use of fats, heavy meats, sweets, starches, and alcoholic beverages must be restricted. Next to diet, regular exercise is the most important therapeutic measure; the patient must eat less or burn up more material, and the chief stimulant of tissue-metabolism is exercise. A change of scene may effect brilliant results. Frequent bathing with salt water followed by friction is a valuable adjunct. When the gastric digestion is weak, mineral acids, strychnine, and pepsin are useful remedies. The salts of potassium and lithium are solvents of uric acid; citrate of lithium (gr. xx), benzoate of lithium (gr. v), or citrate of potassium (gr. xx), may be given, well diluted, two hours after meals. Mineral-waters containing these salts may be recommended. The bowels should be kept regular by some simple laxative.

DIABETES.

(Diabetes Mellitus.)

DEFINITION.—A nutritional disease, characterized by the persistent presence of sugar in the urine, polyuria, and loss of flesh and strength.

ETIOLOGY.—Heredity, adult life, male sex, the Hebrew race, prolonged mental anxiety, and dietetic errors are predisposing causes. It rarely follows injury of the brain or cord.

PATHOLOGY.—The lesions found after death have been so varied that the condition which is really responsible for diabetes is still undetermined. Puncture of the floor of the fourth ventricle will produce glycosuria, but the cases are rare in which lesions of this region have been found after death. In a notable number of cases the pancreas is the seat of cirrhosis and of fatty degeneration, but the relation of this condition to diabetes is still unknown. The liver is frequently enlarged and the seat of degeneration changes. The kidneys are enlarged and often reveal evidences of parenchymatous inflammation.

According to one view, diabetes has its origin in the sympathetic nervous system, and results from a vaso-motor dilatation of the hepatic vessels causing a disturbance of the glycogenic function of the liver and the discharge of glucose in the urine.

According to another theory, diabetes results from a functional or organic disease of those organs, particularly the pancreas and liver, which are engaged in the assimilation of starches and sugars.

SYMPTOMS. *Urinary Phenomena.*—The urine is increased in quantity, the amount varying from three or four pints to as many gallons; its color is pale; its specific gravity ranges from 1015 to 1050; it has a sweetish taste and an aromatic odor. In summer it attracts flies and rapidly ferments. It may leave a whitish residue on the clothes. The percentage of glucose varies from a half per cent. to ten per cent.; the total amount excreted in twenty-four hours varies from a few ounces to a pound or more.

General Phenomena.—There is loss of flesh and strength; the temperature is normal or subnormal; the appetite is often inordinate, and the thirst unquenchable; the tongue is

often fissured and beefy-red; the bowels are usually constipated. The muscles are sometimes the seat of painful cramps.

Cutaneous Phenomena.—The skin is harsh and dry, and frequently the seat of intense itching. Pruritus is especially observed at the genitalia, and this may be the first subjective symptom.

Nervous Phenomena.— Headache, depression of spirits, diminished or lost patellar reflexes, impaired sexual power, dimness of vision, and neuralgia.

The duration varies from a few weeks in the acute form to many years in the chronic form.

Complications. — Pulmonary tuberculosis, pneumonia, gangrene of the lung; defective vision from soft cataract, retinitis or atrophy of the optic nerve; cutaneous lesions, as boils, eczema, carbuncles, and gangrene; nephritis; neuritis and diabetic coma, or *acetonæmia.*

This last condition is characterized by epigastric pain, dyspnœa, a fruity odor of the breath, headache, delirium, stupor, and coma; it probably results from the presence of diacetic and oxybutyric acids in the blood.

Diagnosis.—Care must be taken to distinguish *simple glycosuria* from diabetes. The former is recognized by being transient, and unassociated with the constitutional symptoms of diabetes.

Pruritus and apparently causeless loss of flesh and strength should lead to a suspicion of diabetes.

Prognosis.—The younger the patient, the stronger the hereditary tendency, the larger the amount of sugar excreted, the less the glycosuria can be controlled by diet alone, the graver the prognosis. On the other hand, when it occurs after middle life in association with a gouty diathesis, and the glycosuria is not pronounced, the prognosis for a long duration is comparatively favorable. Absolute cure is rarely attainable.

Treatment. *Dietetic Treatment.*—Sugars and starches must be restricted. Since the patient's appetite is often inordinate, it is necessary to regulate the quantity and character of those foods which are recognized as admissible. The following foods may be included in the dietary :—

Animal Foods.—Meats of various kinds (except liver), game, light broths and soups, fish, and eggs.

Vegetables.—Celery, lettuce, cauliflower, tomatoes, mushrooms, string-beans, young onions, olives, water-cress, and spinach.

Beverages.—Buttermilk, skim milk, sour wines (Rhine wines), carbonated waters, and coffee and tea without sugar.

Relishes.—Nuts of all kinds (except chestnuts), cream cheese, and pickles.

Bread.—Bread made of gluten, bran flour, or almond flour. It should be borne in mind that all the gluten flours are rich in starch.

Fruits.—Cranberries, sour cherries, limes, lemons, and red currants.

Substitutes for Sugar.—Saccharin and glycerin.

The following foods should be avoided: Liver, oysters, wheat bread, biscuits, pastry, potatoes, beets, carrots, peas, turnips, parsnips, sweet fruits, rice, barley, tapioca, corn-starch, corn-meal, chocolate, cocoa, syrups, preserves, and most liquors.

Hygienic Treatment.—Graduated exercise; frequent bathing with salt water followed by friction; the use of flannel underclothing; plenty of rest and sleep; and, if possible, a change of scene.

Medicinal Treatment.—Tonics like iron, arsenic, strychnine, alcohol, and cod-liver oil are often indicated. The special remedies are opium and its alkaloids—morphine and codeine—bromide of arsenic, ergot, antipyrin, salicylate of sodium, and alkalies. Opium is generally the most useful drug; it should be given in small doses gradually increased until the patient takes five or six grains daily. Codeine (gr. $\frac{1}{2}$ increased to gr. vj a day) has been thought preferable to either opium or morphine, but according to the clinical experiments of Bruce and Osler, morphine is much more reliable. The latter may be employed in doses of one-fourth of a grain three or four times daily. The bromide of arsenic is sometimes of decided value; it may be given in the following solution:—

℞ Liq. arsenici brom., f℥j.
Sig.—Two to five drops well diluted after meals.

In gouty patients a course of Carlsbad water with salicylate

of sodium (gr. iij–v thrice daily) and antipyrin (gr. v–x thrice daily) may be recommended, or :—

℞ Sodii salicylat., ʒiij ;
Liq. potass. arseuitis, fʒj ;
Glycerini, fʒj ;
Aq. cinnamomi, ad fʒiij.—M. (J. C. Wilson.)

Sig.—A teaspoonful to a dessertspoonful thrice daily

Diabetic coma is always fatal, but inhalations of oxygen or the subcutaneous injection of large quantities of normal saline solution at intervals may give a few hours' respite, in which consciousness returns.

DIABETES INSIPIDUS.

Definition.—A chronic condition characterized by the excretion of large quantities of pale, limpid urine of low specific gravity and free from albumin and sugar.

Etiology.—Diabetes insipidus must be distinguished from the simple polyuria observed in chronic renal disease, in certain diseases of the brain, and in some cases of hysteria.

Diabetes insipidus sometimes develops without obvious cause. It is more common in the young, and more males are attacked than females. It is occasionally hereditary. It has been induced by injury and by certain diseases of the brain. Profound emotional disturbance has excited it. Syphilis, overwork, and the free use of cold water when the body has been overheated, are reputed causes.

Pathology.—Little is known of the pathology. The kidneys are frequently enlarged and congested, and the ureters dilated.

The theory which is generally accepted as accounting for the polyuria, is that it is due to a vaso-motor paresis of the renal vessels, which permits a free transudation of liquid.

Symptoms.—The disease may begin insidiously or abruptly; the latter is the rule. *The urine:* The quantity is increased, often as much as eight or ten quarts being excreted in the twenty-four hours ; it is pale, and resembles water ; it has a specific gravity of 1002–1005. The total amount of solids is not diminished. Albumin and sugar are generally absent, though there may be a trace of the latter.

General Symptoms.—Insatiable thirst; good appetite; a harsh, dry skin; a dry tongue; constipation; mental apathy; and emaciation.

Duration.—When unassociated with organic disease the duration may be indefinite.

Complications.—These are much less common than in diabetes mellitus. Cataract, pruritus, boils, and tuberculosis have been observed.

Diagnosis. *Diabetes Mellitus.*—The low specific gravity of the urine and the absence of sugar will serve to distinguish diabetes insipidus from diabetes mellitus.

Interstitial Nephritis.—The presence of albumin, hyaline casts, high arterial tension, accentuation of the aortic second sound, and the cardiac hypertrophy will indicate nephritis.

Symptomatic Polyuria.—The history and a careful physical examination will usually prevent an error in diagnosis.

Prognosis.—Usually unfavorable. A permanent cure is sometimes effected. Death results from exhaustion, or more frequently, from some intercurrent disease.

Treatment.—The hygienic treatment suggested for diabetes mellitus is applicable in this disease. No benefit is derived from cutting off the amount of water drunk. Lemonade and other acid drinks may serve to lessen the amount of liquid consumed.

The remedies recommended are ergot, strychnine, opium, valerian, and nitric acid. Galvanism—one pole applied to the neck and the other to the loins—has given good results. When syphilis is suspected, the mercurials and iodides may be administered freely with good hopes of a successful issue.

℞ Pulv. opii, gr. iv;
Acid. gallici, ʒij.—M. (H. C. Wood.)
Ft. in chart. No. xii.
Sig.—One, three or four times daily.

SCURVY.

(Scorbutus.)

Etiology.—Lack of fresh vegetables and bad hygienic surroundings are the predisposing causes.

Pathology. — The pathogenesis of scurvy is unknown. Fatty degeneration from the anæmia, and widespread ecchymoses are found after death.

Symptoms.—The general manifestations of anæmia, with great weakness; spongy, bleeding gums, fetor of the breath, and loosening of the teeth; subcutaneous ecchymoses, and hemorrhages from the mucous membranes; and finally, a painful, brawny induration of the muscles due to a sanguineous exudation.

An infantile form of scurvy (*Barlow's Disease*) sometimes follows the prolonged use of condensed milk, sterilized milk, or proprietary foods. The characteristic symptoms are: Asthenia, anæmia, immobility of the legs, pseudo-paralysis, extreme tenderness, swelling without pitting, thickening of the bones from subperiosteal hemorrhage, ecchymoses, occasionally spongy gums, and a tendency to epiphyseal fractures.

Prognosis.—Favorable in its earlier stages.

Treatment.—Fresh vegetables and the free use of lemon-juice. Iron in moderate doses. Weak solutions of chlorate of potassium or nitrate of silver may be applied to the bleeding gums. In infantile scurvy good results follow the use of fresh milk, beef-juice, and orange-juice.

HÆMOPHILIA.

(Bleeder's Disease, Hemorrhagic Diathesis.)

Definition.—An hereditary disease, characterized by a tendency to bleed excessively from slight wounds, or even spontaneously.

Etiology.—The great cause is heredity. It is more common in males, but is usually transmitted by females, even by those who are not themselves afflicted.

Pathology.—Unknown. In some instances the arteries have been found smaller than normal, with their walls thin and degenerated.

Symptoms. — The chief symptom is free and persistent bleeding after trivial injury. Spontaneous hemorrhages from mucous membranes of the nose, stomach, bowel, etc., and subcutaneous extravasations are quite common. The only other

symptom is a peculiar inflammation of the joints, resembling rheumatism.

PROGNOSIS.—Unfavorable. Grandidier states that one-half die before the eighth year, and less than one-eighth survive their twenty-first. In some instances the tendency is outgrown.

TREATMENT.—Protective and palliative. The bleeding will demand the application of cold compresses and styptics, and the internal use of hæmostatics like ergot, hamamelis, or erigeron. The resulting anæmia will be benefited by iron.

PURPURA HEMORRHAGICA.

(Morbus Maculosus Werlhofii.)

DEFINITION.—A condition arising without obvious cause, and characterized by extravasation of blood in the skin and bleeding from the mucous membranes.

ETIOLGGY.—Bad hygiene, early life, and female sex exert some predisposing influence; but it may occur at any age and in the most robust of either sex. A microörganismal cause has been suggested.

PATHOLOGY.—Unknown.

SYMPTOMS. — The onset may be marked by some fever, headache, malaise, and pain in the limbs; but these symptoms may be absent, and the disease ushered in with a copious crop of small hemorrhages into the skin, followed by bleeding from the mucous membranes. Anæmia and its associated phenomena develop in severe cases.

DIAGNOSIS.—The absence of high fever and nervous symptoms will separate it from *typhus fever* and *cerebro-spinal meningitis*. The history and the absence of spongy gums and of brawny induration of the muscles will separate it from scurvy. Previous health and the absence of hereditary tendency separate it from *hæmophilia*.

PROGNOSIS.—Depends on the severity. Mild cases recover in from one to two weeks; severe cases may prove fatal in a few days from exhaustion or hemorrhage into the brain. Relapses are common.

TREATMENT. — Rest. Light, nutritious food. Arsenic, iron, turpentine, and the fluid extract of hamamelis are the most serviceable remedies.

DISEASES

OF THE

NERVOUS SYSTEM.

DISTURBANCES OF MOTION.

These consist, for the most part, of loss of power, or paralysis, and manifestation of motor excitation, such as convulsions, choreiform movements, and tremors.

Paralysis.

The paralysis may be irregularly distributed, or it may involve a single member, when it is termed *monoplegia;* a lateral half of the body, when it is termed *hemiplegia;* or the body from the waist down, when it is termed *paraplegia.*

Irregular paralysis may result from :—

1. Disseminated lesions in the motor areas of the brain, which are commonly syphilitic.

2. Lesions in the basal ganglia—pons, crura cerebri, medulla, when it is often associated with headache, vomiting, vertigo, and optic neuritis.

3. Acute poliomyelitis. This develops abruptly; it occurs in young children; and it is followed by rapid improvement in some muscles and permanent atrophy and paralysis in others.

4. Chronic poliomyelitis. This develops in middle life; begins in the small muscles of the hand; is associated with atrophy; and progresses very slowly.

5. Idiopathic muscular atrophy. This commonly develops during adolescence; involves the muscles of the arm, shoulder,

buttocks, and thigh; is associated with atrophy; and can be frequently traced to heredity.

6. Pseudo-muscular hypertrophy. This develops in children; is associated with enlargement of the affected muscles; and can be frequently traced to heredity.

7. Multiple neuritis. This is recognized by the history, pain, disturbances of sensation, and tenderness over the nerve-trunks.

8. Syringo-myelia. This is rare; develops during adolescence; and is recognized by pains, atrophy of the affected muscles, a spastic condition of the paralyzed members, and a loss of thermic and painful sensations, while tactile sensation is retained.

Monoplegia may result from :—

1. A focal lesion in the cortical area of the brain. This may be recognized by the history, the absence of wasting, of sensory disturbances, and of the reactions of degeneration.

2. A lesion of the peripheral nerve, from traumatism, neuritis, or the pressure of a tumor. Brachial monoplegia frequently results from the pressure of the head on the arm during sleep. Monoplegia of peripheral origin is recognized by the history, the wasting, the sensory disturbances, and the presence of reactions of degeneration.

3. Hysteria. This may be recognized by the history, sex, and temperament; the paroxysmal character of the paralysis; the disturbances of sensation; and contractures without atrophy or electrical disturbances.

Facial monoplegia may result from a small lesion in the facial centre of the cortex or in the medulla; or from involvement of the nerve in the canal of the temporal bone; or after its exit from the stylo-mastoid foramen.

Facial diplegia (double facial paralysis) generally results from a lesion at the base of the brain.

Hemiplegia may result from :—

1. A diffuse lesion of the motor cortex. The paralysis is on the opposite side of the body and is unassociated with anæsthesia.

2. A lesion of the internal capsule or the adjacent ganglia (corpus striatum and optic thalamus). This is the most

common seat of hemorrhage; the paralysis is on the opposite side of the body and is unassociated with anæsthesia.

3. A lesion of the crus cerebri. This frequently produces hemiplegia and hemianæsthesia on the opposite side, and paralysis of the oculo-motor nerve on the side of the lesion, indicated by dilated pupil, strabismus, and ptosis.

4. A lesion of the pons. This frequently produces hemiplegia and hemianæsthesia on the opposite side, and facial paralysis on the side of the lesion.

5. A lesion in the medulla. This is rare, and is associated with paralysis of the cranial nerves, difficult articulation, cardiac and respiratory disturbances, and vomiting.

6. A unilateral lesion high in the cord (very rare). This produces a spastic paralysis on the side affected, and hemianæsthesia on the opposite side ("Brown-Séquard's paralysis").

7. Hysteria. This may be recognized by the history, sex, and temperament; by being frequently paroxysmal; by its association with sensory disturbances; by the absence of wasting and of abnormal electrical reactions; and by the escape of the facial muscles.

Paraplegia may result from:—

1. Hemorrhage into the cord at the dorsal region. The paralysis develops abruptly, and is associated with complete anæsthesia and involvement of the bladder and rectum.

2. Hemorrhage into the membranes of the cord. The paralysis develops rapidly, but more slowly than the preceding; is associated with intense tearing pains and incomplete anæsthesia.

3. Some forms of multiple neuritis. This is recognized by the pains, the disturbances of sensation, the tenderness over the nerve-trunks, and the absence of "girdle pain" and sphincter involvement.

4. Fracture of the vertebræ.

5. Acute myelitis. The paralysis develops in the course of a few days, and is associated with anæsthesia, bedsores, involvement of the bladder and rectum, loss of reflexes, and wasting of the muscles.

6. Landry's disease (acute ascending paralysis). This develops in the course of a few days, and is unassociated with

anæsthesia, wasting of the muscles, bedsores, or sphincter involvement.

7. Chronic myelitis. This develops over several years, and is associated with numbness and tingling, increased reflexes, involvement of the bladder and rectum, and anæsthesia.

8. Compression of the cord from morbid growths, aneurism, or spinal caries. This may be recognized by the history, the symptoms of the primary disease, the anæsthesia or hyperæsthesia, and the intense pains radiating along the line of the spinal nerves.

9. Lateral sclerosis. This develops slowly and is associated with a spastic condition of the muscles and with increased reflexes, and lacks sensory disturbances.

10. Injury of the brain in delivery (spastic paraplegia of infants). The symptoms resemble lateral sclerosis, and are often associated with imbecility or idiocy.

11. Hysteria. This may be recognized by the history, sex, and temperament; by being frequently paroxysmal; and by the absence of wasting and of abnormal electrical reactions.

12. Caisson disease (divers' paralysis). The history will establish the diagnosis.

Convulsions.

A convulsion is a condition in which there are excessive muscular contractions, continued or intermittent, dependent upon an involuntary discharge of motor impulses from the nerve-centres.

Intermittent contractions are termed *clonic;* continued contractions, *tonic.*

Convulsions may be general or local. The term *spasm* is sometimes applied to the latter.

Varieties of Convulsions.—Three varieties are frequently made: (1) Epileptiform; (2) tetanic; (3) hysteroidal.

Epileptiform Convulsions.—In this form there is unconsciousness, and the movements are for the most part clonic. Epileptiform convulsions may result from :—

1. Idiopathic epilepsy. This condition usually develops before puberty, and the convulsions are general and are unassociated with any definite cause.

2. Organic brain disease. In this condition there may be a history of syphilis or of injury; the convulsions may be local, or begin as such and become general; and there may be concomitant symptoms of cerebral disease.

3. Toxic agents in the blood. Alcoholism, the infectious fevers, and uræmia are frequently associated with convulsions.

4. Reflex irritation. Such convulsions are usually observed in young children, and result from gastric irritation, an adherent prepuce, intestinal parasites, or teething. Convulsive seizures sometimes result from the injection of substances into the pleural sac for the cure of hydrothorax.

5. Cerebral anæmia. Such convulsions are seen after profuse hemorrhage, in fatty heart, and in poisoning from cardiac paralyzants like aconite and veratrum viride.

Eclampsia. This term is applied to designate accidental convulsions, such as the convulsions of childhood resulting from reflex irritation, and the convulsions of pregnancy or the puerperium, resulting from toxic materials retained in the blood.

Tetanic Convulsions.—In this form the discharges emanate from the spinal cord, and are not associated with a loss of consciousness. Tetanic convulsions may result from :—

1. Tetanus. This is recognized by the history of a wound, the tonic character of the convulsions, the early involvement of the jaw, and the absence of fever.

2. Spinal meningitis. This is recognized by exquisite pain in the back, fever, and late involvement of the jaw.

3. Strychnia-poisoning. This is recognized by the history, the intermittent character of the convulsions, the absence of fever, and the escape of the muscles of the jaw until very late.

4. Tetany. In this condition the extremities are chiefly involved; the convulsions are intermittent, and can be produced by pressure on the nerves and arteries of the affected limbs.

Hysteroidal Convulsions.—These are manifestations of hysteria, and in them consciousness is only partially or apparently lost. They are not preceded by an aura, but sometimes by a

sensation of a ball in the throat—the "globus hystericus;" the eyes are partially closed; the face expresses some emotion; the tongue is not bitten; the movements are tonic, or if clonic, appear wilful; the paroxysm is of long duration; and the patient frequently weeps or laughs.

Local Convulsions or Spasms.—*Spasm of the face* may result from a (1) cortical lesion in the inferior portion of the ascending frontal convolution; (2) from *tic convulsif*—a condition occurring in young children, affecting the facial and neighboring muscles, and associated with mimicry, a tendency to use profane language, and various mental disturbances; (3) from habit (habit chorea); and sometimes from (4) *tic douloureux*—neuralgia of the fifth nerve.

Temporary spasms of one arm or one leg are usually manifestations of Jacksonian epilepsy (focal epilepsy), but they sometimes result from hysteria.

Spasm of the hand developing when the member is put to use may result from writers' cramp, Thomsen's disease, or hysteria.

Spasm of the cervical muscles (wry-neck, torticollis) may result from congenital shortening of the sterno-mastoid, myalgia, hysteria, caries of the vertebræ, or the irritation of enlarged cervical glands.

Spasms of the larynx, œsophagus, and diaphragm (hiccough) have already been discussed.

Saltatory Spasm.—This term is employed to designate a condition allied to hysteria, in which a violent spasm seizes the muscles of the leg as soon as the feet touch the ground, and as a result the patient is thrown violently into the air.

Salaam Convulsions.—These consist of violent paroxysmal bobbing movements of the head or trunk, and may be associated with hysteria, chorea, or rarely, organic brain disease.

Choreiform Movements.

These are coarse, jerky, irregular, involuntary movements which more or less simulate purposive movements. They may result from :—

1. Idiopathic chorea (St. Vitus's dance). This disease is

seen in children; is usually mild; runs a course of several weeks; and is prone to be followed by endocarditis.

2. Chorea insaniens. A grave disease occurring in adults, especially pregnant women, and characterized by violent movements, delirium, and fever.

3. Huntingdon's chorea (chronic chorea). An affection occurring in adult life, generally hereditary, and characterized by irregular movements, disturbance of speech, and increasing dementia.

4. Organic brain disease. Choreiform movements are frequently observed in cerebral palsies of children; they may also develop on one side of the body before an attack of apoplexy (pre-hemiplegic chorea), or in the paralyzed members after the hemorrhage (post-hemiplegic chorea).

5. Peripheral irritation. Choreiform movements sometimes develop in pregnancy, and are occasionally noted in stumps after amputation.

6. Habit. Children frequently acquire, through constant repetition or mimicry, choreiform movements which may last indefinitely.

7. Hysteria. The marked rhythmical character of the movements and the history will aid in the recognition of hysterical chorea.

8. Disseminated cerebro-spinal sclerosis. This disease usually induces tremors, but not uncommonly the movements are choreiform. The increased reflexes, the nystagmus, the loss of power, the spastic gait, and the impairment of intellect will aid in its recognition.

9. Paramyoclonus multiplex. A very rare disease, of unknown origin, characterized by continued or paroxysmal choreiform movements which develop or increase under excitement or muscular effort.

Athetosis.

This term was employed by Hammond to designate certain movements occurring chiefly in the hands and feet, and characterized by slow twisting, intertwining, separation, and extension of the fingers and toes. Athetosis is frequently observed

in the cerebral palsies of children, and it occasionally occurs in adults as a result of lesions in the basal ganglia.

Tremors.

A tremor is a fine vibratory movement due to the alternate contraction and relaxation of antagonistic muscles. Tremors are observed in the following conditions:—

1. They may exist from birth unassociated with other symptoms.
2. They may depend upon a lowered tone of the nervous system, being frequently observed in neurasthenia and in the convalescence from acute disease.
3. They may be toxic, resulting from alcoholism or mercurial poisoning.
4. They may be due to old age.
5. They are frequently a symptom of organic disease of the brain and cord; as such, they are met with in paretic dementia, and especially in disseminated sclerosis.
6. They may be the chief symptom in paralysis agitans.
7. They may be hysterical.

The Gait.

The Ataxic Gait.—In locomotor ataxia the patient raises the foot high, throws it forward, and brings it down suddenly, so that the whole sole comes in contact with the floor at once.

Spastic Gait.—In spastic paraplegia the movements are stiff, the knees come together, the leg drags behind, and the toe catches the ground.

Festination.—This term is applied to the gait of advanced paralysis agitans; in walking, the body inclines more and more forward, and the steps grow faster and faster until the patient falls, straightens himself by an effort, or finds support in some neighboring object.

Steppage Gait.—In chronic multiple neuritis the patient raises the foot high, turns the toe up, and brings the heel down first.

The Gait of Pseudo-muscular Hypertrophy.—The feet are wide apart, the belly protrudes, and the movements are clumsy and waddling.

Titubation.—This term is applied to the peculiar gait observed in lesions of the cerebellum. It resembles the gait of locomotor ataxia, but is much more staggering. It is not dependent upon loss of coördination, for in lying down the patient can perfectly control his movements. The absence of the Argyll-Robertson pupil, of sharp pains, and of diminished reflexes will separate cerebellar disease from locomotor ataxia.

The Reflexes.

The Knee-jerk, or Patellar Tendon Reflex.—This is obtained by tapping the quadriceps tendon between its insertion and the patella while the leg is crossed over its fellow.

The knee-jerk is increased in the following conditions :—

1. Frequently in organic disease of the brain, probably from irritation of the cord.
2. In lesion of the cord above the lumbar enlargement, probably from cutting off the influence of the reflex inhibiting centre in the upper part of the cord.
3. In disseminated cerebro-spinal sclerosis and in lateral sclerosis.
4. In irritability of the cord, as in mania, hysteria, strychnia-poisoning, and spinal meningitis.

The knee-jerk is diminished or absent in the following conditions :—

1. Degeneration of the muscle, as in pseudo-muscular hypertrophy.
2. In lesions of the nerves which cut off the impulse from the cord—as neuritis.
3. In lesion of the posterior columns of the cord, as in locomotor ataxia.
4. In poliomyelitis, acute and chronic (the anterior gray matter is part of the reflex centre).
5. In advanced myelitis, when the cord is sufficiently injured.
6. In exhaustion of the spinal centres, as after prolonged laborious work.
7. In poisoning from drugs which depress the cord, as antimony, chloral, etc.

Ankle-clonus.—This consists of vibratory movements obtained by supporting the *tendo-Achilles* with one hand, while the foot is strongly flexed with the other. It can rarely be obtained in health, but is often marked in hysteria and in lateral sclerosis.

Arm-jerk.—This is obtained by striking the biceps tendon at the elbow, or the triceps tendon above the olecranon.

Jaw-jerk.—This is obtained by tapping the jaw while the mouth is partially open.

The Superficial Reflexes.—These are probably true reflexes, and consist in muscular contractions resulting from irritation of the skin.

The following table is based upon the description given by Ross in his Handbook of Nervous Diseases :—

The Reflex.	Produced by	Depends upon Integrity of
Plantar . . .	Tickling the sole of the foot.	The lower end of the cord (conus medullaris).
Gluteal . . .	Stimulating the skin over the buttock.	Loops through the fourth and fifth lumbar nerves.
Cremasteric .	Stimulating the skin on the inner side of the thigh.	First and second pairs of lumbar nerves.
Abdominal . .	Stroking the skin on the side of the abdomen.	The arcs from the eighth to the twelfth dorsal nerves.
Epigastric . .	Stimulating the sides of the chest in the fifth and sixth intercostal spaces.	The arcs from the fourth to the seventh pairs of dorsal nerves.
Erector Spinal	Irritation from the angle of the scapula to the iliac crest.	The arcs in the dorsal region of the cord.
Scapular . .	Irritation of the scapular region.	The arcs of the upper two or three dorsal and the lower two or three cervical nerves.
Palmar . . .	Tickling the palm.	The arcs through the greater part of the cervical enlargement.

The chief cranial reflexes are contraction of the palatal muscles by irritation of the fauces; sneezing, by irritation of the nares; cough, by irritation of the larynx; closure of the eyelids, by irritation of the conjunctiva; and contraction of the iris, by light.

Paradoxical Contraction. (Westphal.)—This is a peculiar phenomenon consisting of a tetanic contraction of the tibialis anticus, lasting for several minutes, and induced by forcibly flexing the foot on the leg. Its cause is unknown. It has been observed in early locomotor ataxia, multiple sclerosis, hysteria, and paralysis agitans.

DISTURBANCES OF SENSATION.

These consist chiefly in a loss of sensation—*anæsthesia;* increased sensation—*hyperæsthesia;* certain abnormal sensations —*paræsthesia;* and subjective painful sensations—*neuralgia.*

Anæsthesia.

Ordinary cutaneous sensibility may be tested by the prick of a pin, by a pinch, or by the faradic current.

Anæsthesia results from interruption of the sensory tract in the nerves, as by neuritis; from interruption of the sensory tract in the cord or brain; from organic disease of the sensory area of the brain; from the action of toxic substances on the nerves or centres; from certain functional conditions like hysteria; and from reflex irritation.

Hemianæsthesia.—A loss of sensation on a lateral half of the body. It may result from :—

1. Hysteria. This is often unassociated with paralysis of motion, and may be recognized by the history, sex, and temperament of the patient; by the paroxysmal character of the anæsthesia; and by exclusion of other causes.

2. A unilateral lesion high in the cord. This is very rare, and may be recognized by being associated with hemiplegia on the opposite side.

3. A lesion of the medulla (very rare). The hemianæsthesia is usually associated with hemiplegia, paralysis of the cranial nerves, difficult swallowing, and cardiac and respiratory disturbances.

4. A lesion in the pons. The hemianæsthesia is often associated with hemiplegia on the same side, and facial palsy on the opposite side.

5. A lesion in the crus, or peduncle. The hemianæsthesia is often associated with hemiplegia on the same side and paralysis of the oculomotor nerve on the opposite side.

6. A lesion of the posterior limb of the internal capsule, or of the optic thalamus pressing on the capsule.

7. A lesion of the occipital cortex.

Monanæsthesia.—A loss of sensation in one member. It may result from hysteria, from a focal lesion of the occipital cortex, or from a lesion of the nerves supplying the member.

Paranæsthesia.—A loss of sensation in all parts below the waist. It may result from hysteria, organic diseases of the cord, neuritis of the lower extremities, or reflex irritation.

Thermo-anæsthesia.—Insensibility to heat or cold occurring as an independent condition. It is sometimes observed in hysteria and syringo-myelia.

Analgesia.—Insensibility to pain. It is sometimes observed in hysteria, in syringo-myelia, and in lesions of the spinal cord.

Retardation of Sensations.—This is frequently observed in all forms of anæsthesia, but especially in the anæsthesia of locomotor ataxia.

The Sense of Space.—The distance at which two points of contact can be recognized as two points. Normally the distance varies in different parts and in different individuals.

On the cheek it is 11–15 millimeters.

On the forehead, 22 millimeters.

On the forearm, 40 millimeters.

On the chest, 45 millimeters.

On the thigh and upper arm, 68 millimeters.

On the leg, 40 millimeters.

On the palm of the hand, 8–12 millimeters.

On the back of the hand, 31 millimeters.

Hyperæsthesia is increased sensibility to external impressions.

It is commonly observed in hysteria, especially in connection with the joints, breasts, genitalia, and spine. It is also observed in neurasthenia, and in beginning inflammation of the nerves and of the cerebro-spinal meninges.

Paræsthesia.—This term is used to indicate certain disagreeable subjective phenomena, such as numbness, tingling, itching, creeping, and "pins and needles."

Paræsthesia is observed in many conditions, as hysteria, spinal sclerosis, neurasthenia, and injury or inflammation of the nerves.

Girdle Sensation.—The sense of having a girdle or tight band around the trunk. It is frequently observed in spinal sclerosis.

Neuralgia.—This consists of paroxysms of severe pain radiating along the line of the nerve-trunks. The pain is relieved by pressure, but there are tender spots (*points douloureux*) where the nerve makes its exit from bony canals or muscular coverings.

Lightning-pains.—This term is applied to the sharp lancinating pains observed in locomotor ataxia. They usually occur in the extremities, and may be mistaken for rheumatism.

Causalgia.—This term has been applied by S. Weir Mitchell to an intensely burning sensation generally observed in "glossy skin."

Pressure Sense.—By this sense the amount of pressure exerted on a given part of the body is determined. It may be tested by placing upon the palms or fingers objects of the same bulk but of different weight, the hands being supported upon a table.

Muscular Sense.—This is the sense by which weight, muscular effort, and position are determined. It is often defective in hysteria, locomotor ataxia, and in many forms of paralysis.

DISTURBANCES OF NUTRITION.

These consist in atrophy of the muscles, changes in electro-muscular contractility, tissue-metamorphoses, and in certain abnormalities of the appendages.

Muscular Atrophy.

Atrophy, or wasting of the muscles results from:—

1. Inactivity. Cerebral palsies may thus be associated with *slow* wasting.

2. Lesions of the cells in the anterior gray horns of the cord, as in acute and chronic poliomyelitis.

3. Lesions of the nerves, such as neuritis or traumatism.

4. Certain diseases of the muscles themselves, as idiopathic muscular atrophy.

The atrophy which attends chronic affections of the joints probably results from neuritis.

The Reaction of Degeneration.

In muscular paralysis there may be simply diminished electrical excitability. This is termed a *quantitative* change. In some cases, however, there is a complete reversal of the normal phenomena. This is termed a *qualitative* change, or the *reaction of degeneration.*

The reactions of degeneration are obtained with the *galvanic current* applied to *muscles* in the *advanced stage of degeneration.*

The subjoined table, setting forth the electro-muscular phenomena in health and disease, follows closely the description of H. C. Wood:—

The anode—the positive pole; the cathode—the negative pole. When a galvanic current of moderate strength is employed, and the cathode is placed over the normal muscle, a strong contraction occurs when the circuit is closed; when the anode is placed over the muscle the contraction is much less; in neither case is there any contraction when the current is broken. When a strong current is used contractions are produced, and the anodal contraction is greater than the cathodal. The reaction of degeneration consists in a reversal of these phenomena.

Normal muscle.

Anodal closing contraction (AnClC) is less than the cathodal closing contraction (CaClC).

Anodal opening contraction (AnOC) is greater than the cathodal opening contraction (CaOC).

Muscle in first stage of degeneration.

Anodal closing contraction (AnClC) equals the cathodal closing contraction (CaClC).

Anodal opening contraction (AnOC) equals the cathodal opening contraction (CaOC).

Muscle in advanced stage of degeneration.

Anodal closing contraction (AnClC) is greater than the cathodal closing contraction (CaClC).

Anodal opening contraction (AnOC) is less than the cathodal opening contraction (CaOC).

The reactions of degeneration are observed in diseases which destroy the trophic cells in the anterior gray horns of the cord or which cut off their influence. Thus they are observed in acute and advanced chronic poliomyelitis, in acute central myelitis, in severe neuritis, and after section or compression of the nerves.

Arthropathies.

An arthropathy is a degenerative affection of the joints, characterized by marked swelling due to effusion, erosion of the cartilages, relaxation and calcification of the ligaments, and atrophy of the heads of the bones. Arthropathies are observed especially in locomotor ataxia, syringo-myelia, and in cerebral hemiplegia. Some regard the joint-phenomena of rheumatoid arthritis as belonging to this class.

Ulceration Resulting from Perverted Nutrition.

Acute Decubitus.—This term is applied to ulcers appearing in a few hours or days, on parts subjected to pressure, after the occurrence of a severe cerebral or spinal lesion.

Chronic Decubitus.—This term is applied to the ulcers which ultimately appear on parts subjected to pressure in the course of chronic spinal affections.

Perforating Ulcer of the Foot.—This term is applied to an undermining ulcer of the foot most commonly observed in locomotor ataxia. It frequently penetrates the deep structures and involves the bones.

Symmetrical Gangrene (*Raynaud's Disease*).—This is a gangrenous affection involving the fingers, toes, tip of the nose, or ears. It arises spontaneously, and is probably due to a vaso-motor spasm.

Trophic Affections of the Skin.—Herpes, scleroderma, vitiligo, chloasma, and the "glossy skin" following injuries of the nerve-trunks, are illustrations of this class of trophic phenomena.

Trophic Affections of the Hair and Nails.—After injury of the nerves and in neuritis the nails often become dry, brittle, and cracked. Under similar conditions there may be a loss of hair, an overgrowth of hair, or a change in the color of the hair.

DISTURBANCES OF CONSCIOUSNESS.

Coma.

Coma is a condition of unconsciousness from which the patient cannot be aroused.

Temporary unconsciousness, due to anæmia of the brain, is termed syncope, which may be recognized by the extreme pallor, weak pulse, and feeble heart-sounds.

1. Coma may result from traumatism. This can only be recognized by the history or the local evidence of injury.

2. *Organic Disease of the Brain.*—The most common cause under this head is apoplexy, which may be recognized by the history, the age, the condition of the arteries, and by evidences

of paralysis, such as unnatural relaxation or rigidity on one side of the body, conjugate deviation of the eyes, or a higher temperature in one axilla.

3. *Epilepsy.*—The coma of epilepsy is usually of short duration. It may be recognized by the history, by the bloody saliva, by the presence of scars on the tongue from previous attacks, and by the exclusion of other causes.

4. *Thermic Fever* (*Sunstroke*).—The temperature of the day or of the room in which the patient is found, the extremely high body-temperature, and the absence of other causes will usually prevent an error in diagnosis.

5. *Certain Drugs.*—Under this head come *alcoholism* and *opium-poisoning.* In *alcoholism* the patient can generally be aroused by shouting in the ear, there is the odor on the breath, and there is an absence of other cause.

In *opium-poisoning* the pupils are small, the respirations are slow, the temperature is normal or subnormal; there may be the odor of laudanum on the breath. The diagnosis will be aided by the exclusion of other causes.

6. *Uræmia.*—In this condition there is a urinous odor to the breath; the aortic second sound is accentuated; the urine is scanty and contains albumin; the temperature may be above or below normal; the pupils are usually small, and there is no evidence of other cause.

7. *The Infectious Fevers.*—The history is sufficient to make the diagnosis. Pernicious malarial fever may produce sudden coma, and in this condition the examination of the blood would render a diagnosis possible.

8. *Hysteria.*—The history, age, and sex of the patient, and the absence of other cause will suggest the condition.

9. *Acetonæmia.*—Diabetic coma may be recognized by the history, the sweetish odor of the breath, the glycosuria, and the subnormal temperature.

Trance.

In this condition the patient lies for several days apparently dead, the pulse and respiration being imperceptible. It is usually a manifestation of hysteria.

Somnambulism.

A dreamlike state, in which the patient performs automatically various feats—such as walking, singing, writing, etc. Mild forms, such as talking and walking in sleep, may occur in health. More marked manifestations occur in hysteria and in hypnotism.

Ecstasy.

A condition of apparent insensibility in which the mind is wholly absorbed with a fancy or delusion. It occurs in the hysterical. The dancing mania of the middle ages is a good illustration of it.

Catalepsy.

This term is applied to attacks characterized by a peculiar stiffness of the muscles, and when this is overcome by force the limbs can be placed in unnatural positions, which they retain for a long time. There may or may not be a loss of consciousness and sensation. It is observed in hysteria, hypnotism, in some cases of epilepsy, in some organic diseases of the brain, and in certain forms of insanity—notably katatonia.

DISTURBANCES OF THE SPECIAL SENSES.

The Eye.

Myosis.—Contraction of the pupil occurs in many conditions, notably in locomotor ataxia, paretic dementia, some cases of disseminated sclerosis, meningitis, cerebral tumor, old age, uræmia, and opium-poisoning.

Mydriasis.—Dilatation of the pupil is also observed in many conditions, notably in atrophy of the optic nerve, paralysis of the third nerve, collapse, severe pain, epileptic seizures, hysterical attacks, belladonna-poisoning, and in some cases of locomotor ataxia and paretic dementia.

Inequality of the Pupils.—This may occur in health, in ocular defects, in organic brain disease, in paretic dementia, in locomotor ataxia, in aneurism pressing on the cervical sympathetic, and in unilateral paralysis of the oculo-motor nerve.

Argyll-Robertson Pupil.—This is one which fails to respond to light, but still accommodates for distance. It is noted especially in locomotor ataxia and paretic dementia.

Conjugate Deviation of the Eyes.—This term is applied to the rotation of both eyes away from the median line. It is noted especially in apoplexy and in convulsions of organic brain disease.

Nystagmus (*Tremor of the Eyeball.*)—It may be congenital, associated with certain ocular troubles, or due to disease of basal ganglia. It is especially frequent in disseminated sclerosis and Friedreich's ataxia.

Optic Neuritis, or Papillitis.—An inflammatory affection of the intraocular end of the optic nerve. The term "choked-disk" is used to designate the condition when it is accompanied with marked swelling. Its chief causes are: Tumor of the brain, cerebral meningitis, syphilis, toxic agents (lead and alcohol), infectious fevers, anæmia, and Bright's disease.

Atrophy of the Optic Nerve.—As a primary affection it is most commonly observed in locomotor ataxia and paretic dementia. Secondary atrophy results from pressure of tumors, aneurisms, etc., on the optic chiasm. Consecutive atrophy is a sequel of optic neuritis.

The Ear.

Tinnitus Aurium (*Noises in the Ear*).—They are observed in cerebral hyperæmia and anæmia, in diseases of the ear, in Ménière's disease, and after the use of certain drugs like quinine and salicylic acid.

Hyperacusis of Hearing.—This is sometimes observed in hysteria, in facial paralysis, and in cerebral hyperæmia.

Deafness generally depends upon disease of the ear itself.

PSYCHICAL DISTURBANCES.

Delusion.—A delusion is a faulty belief concerning a subject capable of physical demonstration, out of which the person cannot be reasoned by adequate methods for the time being. (Wood.)

A *systematized delusion* is one which the patient endeavors to defend by a process of reasoning more or less logical. Systematized delusions are especially observed in monomania.

An *unsystematized delusion* is one which the patient makes no attempt to justify; he asserts his belief without reason. The majority of delusions are unsystematized; and as such are observed in most forms of insanity.

A *fixed delusion* is one which the patient retains for a considerable length of time; it is frequently systematized. Fixed delusions are observed in monomania, paretic dementia, hysterical insanity, and sometimes in melancholia.

An *expansive delusion*, or a *delusion of grandeur*, is one which exalts its possessor. The patient conceives that he is some noted personage, that he is worth millions of dollars, or that he is capable of performing certain marvellous feats. Expansive delusions are frequently observed in paretic dementia, mania, and hysterical insanity.

A *hypochondriacal delusion* is one which depresses its possessor. The patient believes that he has committed the unpardonable sin, that he is being persecuted, or that he is the victim of some dread disease. Hypochondriacal delusions are frequently observed in melancholia, alcoholic insanity, and in some cases of monomania and paretic dementia.

Illusion.—An illusion is a perverted perception. Thus in delirium tremens the patient may transform every piece of furniture into a demon or reptile.

Hallucination. — An hallucination is a false perception, entirely subjective, and not based upon any knowledge derived from without. An individual who hears voices and sees objects when none exist is the subject of hallucinations.

Imperative Conception.—A conception which the person knows to be false, but which, nevertheless, dominates his thoughts and often directs his actions. When he fails to recognize the falsity of his conception, it becomes a delusion.

A morbid impulse is an irresistible desire to commit an act which the patient knows to be wrong. It is usually the result of an imperative conception.

Kleptomania is a morbid desire to steal. *Pyromania* is a morbid desire to set fire to buildings.

Delirium.

Delirium is a mental state characterized by a rapid flight of ideas which are incoherent and often unintelligible. It may result from :—

Acute Delirium (*Bell's Mania*).—A disease arising without obvious cause, and characterized by an abrupt onset, active delirium, a constant repetition of certain phrases or acts, moderate fever, often a bullous eruption, and exhaustion. It generally ends fatally in the course of a few weeks.

Mania.—In this affection the onset is not abrupt. Symptoms of impaired health and mental depression, covering a period of several weeks or months, generally precede the outbreak of the delirium.

Hysteria.—The history, age, sex, and temperament, and the intermittent character of the delirium will aid in the diagnosis.

One of the Infectious Fevers.—Pneumonia and typhoid fever are especially liable to be associated with delirium. The physical signs in the former and the abdominal symptoms in the latter will usually indicate the diagnosis.

Uræmia.—The urinous odor of the breath, the high arterial tension, the accentuation of the second aortic sound, and the presence of albumin and casts in the urine will suggest uræmia.

Alcoholism.—The history, the appearance of the patient, the marked tremors, and frequently terrifying hallucinations will indicate alcoholism.

Inanition.—A form of delirium occasionally arises in the course of exhausting diseases. It is associated with pallor, feeble pulse, and cold extremities. It is generally of short duration, and may be recognized by the circumstances under which it develops.

TUBERCULOUS MENINGITIS.

(Basilar Meningitis, Acute Hydrocephalus.)

DEFINITION.—An acute inflammation of the cerebral meninges excited by the tubercle bacillus.

ETIOLOGY.—In children the disease may be primary, but in adults it is always secondary to a primary focus of tuberculosis in some other part of the body. The majority of cases are observed between the second and the fifth years. Heredity, bad hygienic surroundings, and poor food (milk from a tuberculous mother) are predisposing factors.

PATHOLOGY.—The basilar meninges are especially involved. The pons, crura, and medulla are covered with soft lymph which mats together in a common mass the adjacent nerves and bloodvessels. The tuberculous character of the inflammation is manifested by the presence of small yellowish nodules which are particularly abundant along the bloodvessels in the Sylvian fissures. The amount of fluid in the ventricles is increased, and the ependyma is soft and œdematous. The cortical substance underlying the affected meninges is also soft and infiltrated with leucocytes.

SYMPTOMS.—The disease usually begins insidiously with certain prodromal symptoms. The disposition of the child changes; he ceases to play; he becomes dull and listless, and when disturbed, irritable. Sleep is broken and fitful; the child twitches, grinds his teeth, or starts up with a cry of alarm. Headache develops, and is soon associated with fever and vomiting; the tongue is coated; the appetite lost; and the bowels constipated. When the disease is fully developed the headache becomes intense, and frequently causes from time to time a shrill scream — the "hydrocephalic cry." The special senses are abnormally acute, so that bright lights and loud sounds cannot be tolerated. The surface is also hyperæsthetic, and when touched, the child becomes extremely irritable. The temperature is moderately high (102°–103°); the pulse is at first rapid, but later slow and irregular; the abdominal walls are retracted; the muscles of the neck rigid; and the pupils contracted. Convulsive seizures frequently

develop; they may be general or local. The child lies on one side with the limbs drawn up, the head strongly retracted, and the fingers clinched over the thumb, which is turned into the palm. Towards the close of this stage delirium develops.

When the exudate is sufficient in amount to exert marked pressure, paralytic phenomena develop. Local palsies, especially of the facial muscles, take the place of convulsions; coma follows delirium; the pupils dilate and the eyes roll up; photophobia is replaced by blindness, and intolerance of sound by deafness. If the finger is drawn across the body, a bright red line develops and lingers for some minutes; this is the *tâché cérébrale* of Trousseau. The pulse now becomes rapid and irregular; the respiration assumes the Cheyne-Stokes type, and the temperature falls. The duration is from one to three weeks.

Diagnosis. *Typhoid Fever.*—Typhoid fever may closely simulate meningitis, especially in the young; but the early development of cerebral symptoms, the irregular fever, the slow pulse of the first stage, the retracted abdominal walls, the constipation, and the absence of rose-colored spots will serve to distinguish meningitis from typhoid fever.

Simple Meningitis.—An absolute diagnosis may be impossible, but the history of tuberculosis in the family, the presence of tuberculous foci in other parts, the detection of tubercle on the retina, and an onset without obvious cause will generally indicate the true nature of the case.

Prognosis.—Absolutely unfavorable.

Treatment.—The patient should be placed in a quiet, dark, well-ventilated room. The diet should be liquid. An ice-bag should be applied to the head. Constipation should be relieved by enemata. For the headache, restlessness, and convulsions, chloral and bromide of potassium are useful, and may be given by the rectum.

℞ Moschi, gr. iij;
Camphoræ, gr. xv;
Chloral. hydrat., gr. viiss;
Vitelli ovi, No. i;
Aq. destillat., f℥iv.—M. (Simon.)

Sig.—Wash out the rectum with a simple enema and inject two ounces.

The administration of ergot and of iodide of potassium, and the external application of an ointment of iodoform to the shaved scalp have been recommended, but generally prove useless.

SIMPLE LEPTOMENINGITIS.

(Acute Leptomeningitis, Meningitis of the Convexity.)

DEFINITION.—An acute inflammation of the pia mater not due to tubercle.

ETIOLOGY.—Traumatism, sunstroke, rheumatism, Bright's disease, and the infectious fevers, are the usual predisposing causes. It occasionally develops from caries of the bone which is secondary to middle-ear disease.

PATHOLOGY.—The membranes are opaque, thickened, congested, adherent, and more or less infiltrated with purulent fluid. Generally the convexity is affected, but in some cases, as those following middle-ear disease, the base is chiefly involved. The adjacent cortical substance is also œdematous, soft, and injected.

SYMPTOMS.—Moderate irregular fever, loss of appetite, constipation; intense headache, intolerance to light and sound, contracted pupils, delirium, retraction of the head, convulsions, and coma.

When the base is involved, the symptoms are almost identical with those of tuberculous meningitis.

PROGNOSIS.—Unfavorable, though recovery is not impossible.

TREATMENT.—The patient should be placed in a quiet, dark, well-ventilated room. An ice-bag should be applied to the head. When the patient is robust, wet cups or leeches may be applied to the neck. The diet must be liquid. Constipation should be relieved by enemata. Restlessness, headache, and convulsions call for chloral and bromide of potassium.

CHRONIC LEPTOMENINGITIS.

DEFINITION.—A chronic inflammation of the pia mater.

ETIOLOGY.—It may result from syphilis, alcoholism, traumatism, or sunstroke. It may be secondary to acute infec,

tious leptomeningitis. It is an associated condition in abscess and tumors of the brain.

Symptoms.—Persistent, dull headache, mental deterioration, vertigo, muscular weakness, a low grade of optic neuritis, and occasionally nausea, vomiting, and tinnitus. Acute exacerbations are not infrequent, and are characterized by fever, severe headache, delirium, convulsions, and stupor.

Diagnosis. *Cerebral Tumor.*—In tumor the symptoms are more severe and of a more focal character, and the optic neuritis is of a high grade.

Uræmia.—This condition may be recognized by the albuminuric retinitis and the presence of albumin and casts in the urine.

Prognosis.—More or less unfavorable. A cure is sometimes obtained in cases resulting from syphilis, sunstroke, and traumatism.

Treatment.—In syphilitic meningitis mercury and potassium iodide should be used freely. In other instances courses of ergot and potassium bromide are useful. Applications of the thermo-cautery often give relief. Tonics and hypnotics are frequently indicated.

CHRONIC PACHYMENINGITIS.

Definition.—Inflammation of the dura mater.

Etiology.—Inflammation of the external layer may result from injury, syphilis, sunstroke, or caries of the bone. Inflammation of the internal layer (hemorrhagic pachymeningitis) may be secondary to chronic cardiac or renal disease, one of the infectious fevers, chronic alcoholism, or especially, insanity.

Hemorrhagic Pachymeningitis.

(Hæmatoma of the Dura Mater.)

Pathology.—The membranes are thickened, opaque, and more or less adherent. The bloodvessels are dilated. Between the membranous layers are frequently observed hemorrhagic effusions; these vary in extent from slight ecchymoses to clots as large as a hen's egg. In some cases the pressure of

the clots on the convolutions is sufficient to cause the latter to atrophy.

SYMPTOMS.—Often obscure. In some cases there are no manifestations during life. When the condition is marked, the following phenomena may be observed: Headache, failure of memory, impairment of intellect, stupor, contracted pupils, local convulsions, or palsies. The symptoms may alternately improve and grow worse for a long period. In grave cases, associated with extensive hemorrhagic effusion, the symptoms resemble apoplexy.

DIAGNOSIS.—This can rarely be made with certainty.

PROGNOSIS.—Unfavorable.

TREATMENT.—Grave cases should be treated as apoplexy.

HYDROCEPHALUS.

(Congenital Hydrocephalus, Water on the Brain.)

DEFINITION.—A condition in which there is an excessive accumulation of fluid in the ventricles or arachnoid cavity.

ETIOLOGY.—*Acquired Hydrocephalus* may develop at any period of life, and may result from meningitis, the pressure of a tumor, or from one of the causes of general dropsy.

Congenital Hydrocephalus, the form now under discussion, dates from birth or develops in the first few years of life. Its cause is unknown; in some cases it is probably due to a latent inflammation of the ependyma of the ventricles.

PATHOLOGY.—The head is large and round; the bones are thin and translucent; the sutures and fontanelles are enlarged, and, if life has been prolonged, are filled with numerous Wormian bones. The convolutions of the brain are flattened and the sulci more or less obliterated. In *external hydrocephalus* the accumulation of fluid is found in the arachnoid sac; but in *internal hydrocephalus*—the more common form—the ventricles are greatly distended with a watery fluid of low specific gravity, containing a trace of albumin. The ependyma is often thickened and roughened. Malformations are frequently observed, and probably result from the same cause which induced the effusion.

Symptoms.—Sometimes the disease develops before birth, and the large head interferes with the delivery of the child. In other cases nothing peculiar is observed until the child is several months old, when the swelling of the head attracts the attention of the parents. The head assumes a globular shape; the fontanelles and sutures remain open; the face becomes relatively small; the eyes protrude and are directed downward from the pressure of the fluid on the supraorbital plates; the scalp appears thin and stretched; the superficial veins are distended; and the hair becomes scant. In some cases the head is so heavy that the thin neck can no longer support it, and it falls forward on the breast.

As a rule, the intelligence is considerably impaired, but exceptional cases are marked by precociousness. Motor phenomena are frequently present: the reflexes are exaggerated; one or more of the members may be the seat of a spastic paralysis; convulsions develop in many cases.

The duration varies in different cases. The large majority soon die of inanition, convulsions, or some intercurrent disease to which their reduced vitality makes them an easy prey; but in a few, life is prolonged for many years.

Diagnosis.—Hydrocephalus must not be mistaken for *rachitic enlargement of the head*. In the latter, the head is square instead of globular; the intelligence is good; there are no motor phenomena; and bony enlargements are usually detected at the ends of the long bones and at the junction of the cartilages with the ribs.

Prognosis.—Unfavorable. In a few cases arrest of the disease has been spontaneous, or has resulted from aspiration of the fluid.

Treatment.—The treatment is unsatisfactory. Counter-irritation and the use of diuretics and absorbents exert no influence on the disease. In the majority of cases, beyond dietetic and hygienic measures and the occasional use of tonics, little can be recommended. In cases where the pressure-symptoms are marked, tapping offers some hopes of temporary relief. After the operation compression of the skull should be made by the application of concentric bands of adhesive plaster.

PARETIC DEMENTIA.

(General Paralysis of the Insane, General Paresis, Chronic Meningo-encephalitis.)

Definition.—A chronic inflammatory affection of the cerebral cortex, characterized by a change of disposition, failure of memory, mental exaltation, delusions of grandeur, tremors, epileptiform seizures, and paralysis.

Etiology.—Male sex, middle life, prolonged mental strain, and excesses are predisposing factors. It may be induced by the usual causes of sclerosis, namely, syphilis, alcoholism, lead-poisoning, gout, etc.

Pathology.—The membranes are opaque, thickened, and at places, adherent to the brain substance. The cortex is more or less atrophied and increased in firmness. Microscopic examination reveals an overgrowth of connective tissue and degeneration of nerve-fibres and ganglionic cells.

In some cases similar degenerative changes are observed in the posterior and lateral columns of the cord.

Symptoms.—The disease usually begins insidiously with a change in disposition; the industrious become slothful; the ambitious, apathetic; the chaste, dissolute; the liberal, parsimonious; the complaisant, churlish; and the truthful, false. The energy relaxes, the judgment weakens, and the memory fails. As the faculties become impaired, a peculiar egotism and a mental exaltation develop; the patient becomes boastful, loquacious, and easily provoked to furious outbreaks. The failure of memory is early noted in writing, by the use of wrong letters and the suppression of syllables. At this time motor phenomena may be observed: the tongue trembles when it is protruded; the speech is slow, hesitating, and indistinct; the pupils are often unequal; and the gait is somewhat shuffling.

The most characteristic psychical symptom of fully-developed paretic dementia is the delusion of grandeur: the patient conceives that he is some distinguished personage, that he owns acres of land, or that he is the inventor of some wonderful machine. The mind is usually serene and cheerful,

but periods of depression are not infrequent. The sensibilities are blunted and the animal nature emphasized. The mind becomes more and more involved; there is extreme indifference to all that transpires; the appetite is voracious, and in eating the patient bolts his food and soils his clothes. The tremor of the tongue increases, and spreads to the lips and other parts of the face; the speech is indistinct and "scanning;" the pupils fail to respond to light, but still accommodate for distance (Argyll-Robertson pupil); the reflexes are generally increased. Spells of unconsciousness resembling *petit mal* are not uncommon.

In the final stage mental power is almost obliterated; the health fails; the bladder and rectum become unretentive; the gait is more unsteady; and at last the patient is unable to leave his bed. Death usually results from exhaustion or intercurrent disease.

Diagnosis.—The insidious change in disposition, failure of memory, tremors, Argyll-Robertson pupil, and delusions of grandeur are the diagnostic features.

Cerebral Syphilis.—In this disease the history, the occurrence of convulsions and of partial facial palsies, the absence of delusions of grandeur and of "scanning" speech, and the effect of treatment will usually prevent an error in diagnosis.

Prognosis.—Unfavorable. The course is not uniform; in some cases there are remissions, or lucid intervals, which last several months or years. The average duration is three or four years.

Treatment.—Rest of body and mind. Careful attention to the hygiene. When there is a suspicion of syphilis, iodides and mercurials should be given a thorough trial. As a rule, patients must be removed to asylums.

CEREBRAL PARALYSIS IN CHILDREN.

Definition. — Hemiplegia, diplegia, or paraplegia appearing at birth or in the first few years of life, and usually associated with atrophy and sclerosis of the cerebral cortex, or porencephalus.

Pathology.—After death one of the following conditions is observed: Atrophy and sclerosis of the convolutions; poren-

cephalus (a cystic condition of the cortex); or more rarely, some local obstruction to the cerebral circulation, as from hemorrhage, embolism, or thrombosis. The exciting cause of the porencephalus and sclerosis is still undetermined.

SYMPTOMS.—*In the hemiplegic* variety the onset is sudden, and is frequently attended with fever, convulsions, or coma. After a few hours or days these severe symptoms subside, and the child is left paralyzed on one side of the body. In rare instances the paralysis ultimately disappears and the child is restored to health, but in the large majority of cases it persists and is followed by secondary rigidity. Imbecility, epilepsy, and choreiform or athetoid movements in the affected members are very common sequelæ.

The diplegic or paraplegic form frequently dates from birth, and is characterized by rigidity and loss of power in all of the extremities. The legs suffer more than the arms. Choreiform or athetoid movements are frequently present. Children thus affected are generally idiots or imbeciles. Meningeal hemorrhage, induced by tedious labor or the use of the forceps, appears to be responsible for this variety.

TREATMENT.—During the convulsive stage an ice-bag should be applied to the head, and chloral or bromide administered by the mouth or rectum. The paralysis resists treatment; but subsequent rigidity may be lessened by massage and passive movements, and the deformity by mechanical appliances.[1]

CEREBRAL HYPERÆMIA.

(Congestion of the Brain.)

ETIOLOGY.—*Acute congestion* results from exposure to the sun; from the use of certain drugs, like alcohol and nitroglycerine; from excesssive brain-work; or from some reflex disturbance, as gastric irritation.

Chronic congestion results from some local obstruction to the return of blood from the brain, as by a tumor in the neck; from obstruction to the general circulation, as in chronic heart and lung disease; from the suppression of some habitual dis-

[1] The above description is based upon Osler's elaborate monograph.

charge, as the menstrual flow at the menopause; or from some general cause, such as prolonged anxiety, overwork, excesses, irregular living, etc.

PATHOLOGY.—The vessels of the meninges and of the brain-substance are engorged.

SYMPTOMS. *Acute Form.*—Intense headache; vertigo; intolerance to light and sound; restlessness; tinnitus aurium; and sleeplessness, or sleep disturbed by horrible dreams.

Chronic Form.—Vertigo; dull headache; failure of memory; irritability; inability to concentrate the thoughts; and disturbed sleep. The symptoms grow worse when the recumbent posture is assumed. Ophthalmoscopic examination reveals retinal hyperæmia. In marked cases there may be exacerbations closely resembling apoplexy, in which there is unconsciousness, followed by temporary paresis.

PROGNOSIS.—Depends on the cause; when this can be removed the prognosis is favorable.

TREATMENT. *Acute Congestion.*—The patient should be placed in a darkened, well-ventilated room. The head and shoulders should be slightly elevated. An ice-bag should be applied to the head. Leeches or wet-cups may be applied to the neck. Sedatives like bromide of potassium and aconite are useful. Ergot may be employed for its power to contract the vessels. If there is constipation, it should be relieved by a brisk saline purge.

In *chronic cases* the cause should be ascertained and, if possible, removed. The habits of the patient must be regulated. The diet must be light and nutritious. Constipation must be relieved by diet or by the occasional use of a saline laxative. Sedatives like bromide of potassium and aconite are useful. In the apoplectiform attacks venesection is indicated.

CEREBRAL ANÆMIA.

ETIOLOGY.—General cerebral anæmia as a *chronic affection* may result from cardiac disease, especially aortic stenosis. It may be associated with general anæmia. It may be due to atheromatous obstruction of the arteries.

Overwork, prolonged emotional excitement, irregular living, and excesses are also said to predispose.

As an *acute condition* it exists in syncope and shock; after hemorrhage; after the sudden withdrawal of fluid from the abdominal sac; and after ligation of the carotid artery.

Symptoms. *Acute Form.*—Pallor of the face, vertigo, confusion of ideas, ringing in the ears, dimness of vision, dilatation of the pupil, nausea, and a tendency to yawn. In extreme anæmia there may be convulsions and coma.

The *chronic form* is characterized by vertigo, headache, disturbed sleep, intolerance to light and sound, irritability of temper, failure of memory, inability to concentrate the attention on one subject, a tendency to syncope, and extreme lassitude. The symptoms improve when the patient lies down. Ophthalmoscopic examination reveals pallor of the retina.

Diagnosis.—Cerebral anæmia closely simulates *cerebral congestion*, but in the latter there is no tendency to syncope; the symptoms grow worse when the patient lies down; the ophthalmoscope reveals retinal hyperæmia; the pupils are contracted instead of dilated, and the urine is apt to be decreased.

Prognosis.—Depends on the cause; when this can be removed the prognosis is favorable.

Treatment.—In acute cases diffusible stimulants like nitro-glycerin, ammonia, and strychnia are indicated. In chronic cases the cause should be ascertained, and if possible, removed. When it is due to general anæmia, iron, arsenic, and quinine are useful remedies. When dependent on valvular disease, rest and the use of digitalis, strophanthus, or strychnine are the remedial measures.

CEREBRAL HEMORRHAGE.

(Cerebral Apoplexy.)

Etiology.—The affection is most commonly met with in the old, in whom the bloodvessels are atheromatous, and in the very young, in whom they are naturally weak. All causes which lead to degeneration of the arteries, such as

rheumatism, gout, syphilis, alcoholism, and Bright's disease, predispose to it. Sufferers from chronic Bright's disease are very liable to die of apoplexy on account of the association of cardiac hypertrophy with arterial degeneration. Heredity predisposes, inasmuch as members of certain families are particularly prone to sclerosis of the vessels. The attack may be precipitated by mental or physical excitement, alcoholic excess, or some reflex disturbance, as gastric irritation. In children it may be excited by a paroxysm of whooping-cough or by a convulsion.

Pathology.—In children the hemorrhage is most commonly cortical; in adults it is usually within the brain-mass. The bloodvessels are generally atheromatous, and are sometimes the seat of miliary aneurisms. The clot varies greatly in size; sometimes it is small, merely a capillary oozing; at other times it may fill a hemisphere. Its most common seat is the internal capsule—the motor highway between the optic thalamus and the corpus striatum. In recent hemorrhages the clot is dark and soft, and the surrounding tissue stained and more or less lacerated. If the hemorrhage has not been very copious, the clot loses it color, shrinks, and is finally absorbed, and the damaged cerebral fibres are replaced by proliferated connective tissue, which contracts and forms a scar more or less pigmented with hæmatoidin. In other cases, instead of a scar, a cyst is formed which encloses a clear straw-colored fluid. Large effusions in the motor path may produce secondary changes—either a softening of the cerebral tissue beyond, or a degeneration which travels down the lateral column of the cord on the side opposite the lesion.

Symptoms.—*Prodromal symptoms* indicating cerebral congestion frequently precede the attack; these are headache, vertigo, disturbed sleep, tinnitus aurium; or there is a sense of numbness or weakness on the side which is to be affected. Persistent vomiting sometimes precedes the hemorrhage.

The Attack.—In many cases the patient falls suddenly unconscious without previous warning. The face is flushed; the eyes are injected; the lips are blue; the breathing is stertorous; the pulse is full and slow; the temperature is at first subnormal from shock, but later it is elevated from irritation;

and the urine and feces may be passed involuntarily. Convulsive seizures are not infrequent; they result from irritation transmitted to the undamaged motor regions. Even while the patient is comatose the paralysis can be detected. The head and eyes may be strongly rotated to one side (conjugate deviation); one cheek often flaps more than the other; the pupils may be irregular; any movements which the patient may make are restricted to the sound side; when the affected arm is raised and let fall, it drops lifeless or manifests an unnatural rigidity; and occasionally there is a difference of temperature in the two axillæ. In grave cases the patient does not awake from the coma; the pulse grows feeble; the respirations assume the Cheyne-Stokes type; the reflexes are abolished; mucus collects in the throat and produces a rattling sound; the temperature rises high; and death results after the lapse of a few hours or one or two days.

In some cases the paralysis develops quite gradually and is not attended with unconsciousness.

Subsequent Symptoms.— When the attack does not prove fatal, consciousness is finally restored, and if the hemorrhage is in its usual location, there remains a hemiplegia on the opposite side. In a few hours the affected muscles become rigid from irritation of the motor fibres. This early rigidity is termed primary rigidity; it lasts from a few days to several weeks and has no significance from a prognostic standpoint. The paralysis is rarely a complete hemiplegia; the muscles of the upper part of the face and thorax usually escape, because they are accustomed to act in unison with their fellows on the opposite side, and such muscles are rarely involved in cerebral hemiplegia. When the tongue is protruded, it deviates toward the paralyzed side. The deep reflexes are exaggerated on the affected side. Sensation is unimpaired unless the posterior limb of the internal capsule is also involved, when there is hemianæsthesia with hemiplegia. The gait is peculiar; in walking the patient supports the paralyzed arm, and swings the leg forward by a rotary movement imparted to it by the trunk. When the clot has been small, the paralysis may completely disappear. More frequently recovery is only partial; the power of the facial muscles is usually restored

entirely, and the leg improves more than the arm. In unfavorable cases the muscles again become rigid (secondary rigidity) from a degenerative process travelling down the lateral column of the cord; this condition is indicative of permanent disability. Generally the mental power remains unimpaired, but sometimes the symptoms of cerebral softening gradually develop.

Diagnosis.—The coma of apoplexy must be distinguished from *uræmia, opium-poisoning, alcoholism,* and *sunstroke.* The age of the patient; the condition of the arteries; the evidence of paralysis; the difference of temperature in the two axillæ; and the absence of other cause will usually prevent an error in diagnosis.

Embolism.—This usually occurs in earlier life; it is commonly associated with valvular disease; premonitory symptoms are rarely present; the pulse is more often weak than strong; disturbances of temperature and breathing are less marked.

Thrombosis.—This also produces hemiplegia, but its development is very gradual; unconsciousness is often absent, and temperature and breathing are not much disturbed.

Hemiplegia from other Causes.—Tumors and abscess in the brain may produce hemiplegia, but the latter develops gradually and is usually associated with other cerebral phenomena, such as persistent headache, vertigo, ocular palsies, choked disk, etc.

Hysterical Hemiplegia.—In hysteria the face escapes; there is frequently anæsthesia on the affected side; the gait is peculiar, in that the patient pushes the paralyzed limb instead of swinging it. These features together with the age, temperament, sex, and mode of onset will usually suggest the true cause.

Prognosis.—Always doubtful. Persistent and complete unconsciousness, high temperature, loss of reflexes, and embarrassed respiration are unfavorable phenomena. When the attack does not prove fatal, there is always a probability of subsequent ones, for the etiological conditions still remain.

Treatment. *Prophylaxis.*—Patients predisposed to apoplexy should lead a quiet life, free from mental and physical

excitement. The diet should be nutritious, but easily digestible. Constipation should be relieved by the occasional use of a saline laxative. To secure a free return of the blood from the brain the clothes at the neck should be loose.

The Attack.—The head and shoulders should be slightly elevated, and an ice-bag applied to the head. Croton oil (gtt. j–iij) in a little glycerine or olive oil may be placed on the back of the tongue to secure prompt catharsis. If the pulse is strong, venesection is indicated and should be continued until the pulse softens. Bleeding cannot undo the damage already done, but by relieving cerebral congestion it may prevent a renewed outpouring. On the other hand, when the face is pale and the pulse feeble the hypodermic injection of diffusible stimulants, like ammonia and strychnia, is indicated. When collections of mucus interfere with breathing, the patient should be gently turned on his side and the mucus removed.

To prevent the formation of bedsores the position should be frequently changed, and the parts subjected to pressure thoroughly cleansed.

Subsequent Treatment.—As other attacks are liable to occur, the prophylactic treatment already referred to is applicable here. Iodide of potassium (gr. v–x thrice daily) may be administered with the hope of absorbing the clot. After the lapse of six or eight weeks, faradism, massage, and passive movements should be applied to the affected muscles. Strychnine by the mouth or injected directly into the muscles is often very useful. Even when the paralysis remains, contractures may be prevented to a considerable extent by massage.

OBSTRUCTION OF THE CEREBRAL ARTERIES.

(Embolism, Thrombosis.)

ETIOLOGY.—*Cerebral emboli* may be derived from the valves of the heart in endocarditis; from an atheromatous plate in the aorta; or from a clot in the heart or in the sac of an aneurism. Obstruction from embolism may occur at any

age, but it is far more commonly observed in young adults than at the extremes of life.

Thrombi are clots formed in the vessels, and a weak heart and arterial degeneration are the predisposing factors. They are usually observed in advanced years, but those dependent on syphilitic arteritis frequently occur in early adult or middle life.

PATHOLOGY.—*Emboli* are most frequently found in a branch of the left middle cerebral artery. When the artery obstructed is a large one, the part beyond usually becomes pale and soft; but sometimes it presents the appearance of an infarction and is infiltrated with blood. Subsequently, microscopic examination reveals fatty degeneration of the nervous elements and more or less pigmentation from extravasated blood. If the area affected is small, absorption may follow and scar-tissue be substituted.

Thrombi are usually found in the middle cerebral, basilar, or vertebral arteries, and are followed by similar changes.

SYMPTOMS.—*An embolus* lodging in the middle cerebral artery usually causes abrupt hemiplegia, and frequently aphasia. There may be no prodromes, and consciousness may be preserved during the seizure.

When the basilar artery is obstructed, there may be extensive paralysis on both sides of the body, and later, symptoms of bulbar disease, namely, paralysis of the lips, pharynx, and œsophagus, disturbance of the heart, and Cheyne-Stokes breathing.

In *thrombosis* the symptoms are similar to embolism, but they develop very slowly, and are frequently preceded by prodromes indicating disturbed cerebral circulation, such as headache, vertigo, disturbed sleep, failure of memory, numbness and tingling in the limbs to be affected.

Subsequent Symptoms.—In both embolism and thrombosis, if the artery obstructed has been large, the paralysis persists and symptoms of cerebral softening appear—namely, failure of memory, vertigo, headache, disturbed sleep, great irritability, and finally dementia.

DIAGNOSIS.—Cerebral embolism closely resembles *apoplexy*,

and sometimes it may be impossible to distinguish between the two conditions. The following are the diagnostic features:—

Embolism is generally associated with valvular disease; it commonly occurs in the young; prodromes are frequently absent; aphasia is more common in embolism than in hemorrhage; there is much less disturbance of temperature after embolism than after apoplexy; consciousness is less apt to be lost in embolism than in apoplexy.

Prognosis.—In embolism it is very doubtful; recovery may follow, but often the paralysis remains. In thrombosis there is very little hope of recovery, unless the cause is syphilis.

Treatment.—After obstruction from embolism the patient should be kept at absolute rest for a few days, and subsequently the paralysis treated as after apoplexy. In thrombosis treatment is of no avail, save in syphilitic subjects, when mercurial inunctions should be employed freely and the bichloride given by the mouth.

CEREBRAL SOFTENING.

Definition.—Degeneration of the brain-substance resulting from impaired nutrition.

Etiology.—Local softening may result from obstruction to the circulation by a tumor, embolism, thrombosis, or clot. Extensive softening may result from prolonged cerebral anæmia or congestion. It is most frequently observed in old people in association with atheromatous arteries.

Pathology.—The affected portion is dull white or reddish-yellow, according to the amount of blood-pigment present; and is less firm than the surrounding brain-substance. Sometimes it is so soft that when the brain is cut a creamy fluid flows out. Microscopic examination reveals destruction of the nerve-elements and their substitution by granular débris and fat-drops.

Symptoms.—When extensive the symptoms are: Failure of memory, irritability of temper, vertigo, headache, partial palsies, cutaneous anæsthesia or paræsthesia, delusions, and finally dementia.

Local softening may be manifested by local paralysis.

DIAGNOSIS. *Cerebral Tumor.*—Tumors usually develop in younger subjects; the headache is more severe; choked disk is frequently observed.

PROGNOSIS.—Unfavorable.

TREATMENT.—Palliative.

MORBID GROWTHS IN THE BRAIN.

(Tumors of the Brain.)

ETIOLOGY.—Early adult life, male sex, and perhaps traumatism predispose. Heredity also predisposes to the extent that it favors the development of cancer, gumma, and tubercle.

VARIETIES.—Tubercle, gumma, glioma, aneurism, cysts, sarcoma, and carcinoma are the most common varieties. Less frequently fibroma, psammoma, and lipoma are observed.

PATHOLOGY.—*Tuberculous tumors, or tyromata*, vary in size from a pea to an egg; they may be single or multiple; and are usually observed in the young.

Gumma.—This appears as a round, yellow, caseous mass, and is nearly always on the surface of the brain, into which it grows from the overlying membranes. It is usually met with between thirty and forty.

Glioma.—This tumor is found almost exclusively in the brain. It arises from the neuroglia, and may be soft like brain-substance or firm like fibrous tissue. It is chiefly met with in the young.

Aneurism.—Encephalic aneurism may be single or multiple. Miliary aneurisms of small vessels frequently excite apoplexy. The most common seats of large aneurisms are the middle cerebral, basilar, and internal carotid arteries.

Cysts.—These are usually congenital (porencephalus), or result from hemorrhage, but sometimes they result from the tænia echinococcus (hydatid cyst), or tænia solium (cysticercus cellulosæ).

Sarcoma.—This is usually a circumscribed tumor, and commonly grows from the membranes, periosteum, or bone.

Carcinoma.—This is nearly always secondary and multiple.

SYMPTOMS.—(1) Headache is rarely absent; it may be

localized and associated with tenderness on pressure. (2) Vomiting is a common symptom, especially in tumors of the base of the brain; it is often unassociated with nausea and does not relieve the attending headache. (3) Ocular phenomena, as optic neuritis, or choked disk, optic atrophy, diplopia, hemianopia, blindness, and irregular pupils. (4) Vertigo. (5) Psychical phenomena, as failure of memory, irritability of temper, depression of spirits, and dementia. (6) Symptoms resulting from local pressure, such as local palsies or convulsions, aphasia, and local anæsthesia.

DIAGNOSIS.—This includes: (1) the existence of a tumor, (2) its character, and (3) its location.

The existence of a tumor is determined by the headache, vomiting, optic neuritis, and symptoms of local pressure.

Abscess.—Cerebral tumor must be distinguished from abscess. The latter usually results from traumatism or is secondary to a focus of suppuration in some other part of the body; its progress is more rapid; choked disk is rare; and there is often febrile disturbance.

Chronic Meningitis.—In this affection the symptoms indicate a diffuse lesion; disturbances of temper, memory, and sleep are more marked; and optic neuritis is not frequent.

The Character of the Growth.—This cannot always be determined. The early age, the rapid progress, and the family history may suggest *tubercle.* The early age, slow progress, and mild pressure-symptoms may suggest *glioma.* The history, age, and concomitant symptoms will indicate *syphilis.* The presence of a primary growth will lead to the diagnosis of *cancer.* The presence of a thrill, *bruit,* and marked tinnitus, and the absence of optic neuritis would suggest aneurism.

Location.—The following facts relating to *cerebral localization* will aid in determining the location of the growth.

Motor area. This consists of the ascending frontal and ascending parietal convolutions, and the paracentral lobule which lies along the median fissure. When the tumor irritates the part, convulsion results; when it exerts enough pressure to destroy function, paralysis results.

Central portion of the motor area—spasm or paralysis of one arm.

The lower portion of the motor area—spasm or paralysis of one side of the face.

Paracentral lobule—spasm or paralysis of a lower extremity.

Posterior part of the third frontal convolution (*left side*)—motor or ataxic aphasia.

Anterior portion of the frontal lobes—sometimes psychical disturbances; often no special symptoms.

Temporal lobe, first and second convolutions (*left side*)—word-deafness.

Parietal lobe—sensory disturbances on opposite side of body.

Angular and supramarginal gyri (*left side*)—word-blindness and apraxia.

Occipital lobe—hemianopsia, and sometimes word-blindness and mind-blindness.

Corpus striatum—large lesions produce hemiplegia from pressure on the internal capsule.

Optic thalamus—large lesions may produce hemianæsthesia from pressure upon the posterior limb of the internal capsule, and sometimes hemianopsia.

Corpora quadrigemina—dilatation and immobility of the pupils, loss of coördination, disordered ocular movements, and hemianopsia.

Crus cerebri—hemiplegia on one side, and paralysis of the oculo-motor nerve on the other.

Pons—paralysis of the cranial nerves, and in many cases hemiplegia and hemianæsthesia on one side, and facial paralysis on the other. Bilateral lesions may produce general paralysis.

Internal capsule (*middle third*)—hemiplegia on the opposite side. *Posterior third*—hemianæsthesia on the opposite side.

Medulla—paralysis of the cranial nerves, difficult articulation, cardiac and respiratory disturbances, vomiting, and sometimes hemiplegia.

Cerebellum (*middle lobe*)—staggering gait, vomiting, severe vertigo, headache, double optic neuritis, and tremors or tonic spasms. Paralysis may result from pressure on the pyramidal tracts.

Prognosis.—Always grave. When the tumor is not gummatous, and is not suitable for operative interference,

the prognosis is absolutely unfavorable. The duration is from a few months to several years.

TREATMENT.—Localized cortical growths, which are not malignant or syphilitic, are suitable for operative interference. In cerebral gumma inunctions of mercury should be employed, and mercury and iodide of potassium given by the mouth. In other cases the treatment is palliative. Cold applications to the head, bromides, antipyrin, and morphine are required to relieve pain.

ABSCESS OF THE BRAIN.

(Suppurative Encephalitis.)

ETIOLOGY.—(1) It may be traumatic. (2) It may be secondary to suppurative inflammation of adjacent parts, as caries of the temporal bone following otitis media. (3) It may be secondary to some distant focus of suppuration, as in pulmonary abscess, hepatic abscess, ulcerative endocarditis. (4) It may follow one of the infectious fevers.

PATHOLOGY.—The abscess varies in size from a pea to one large enough to fill an entire hemisphere. The surrounding tissues are hyperæmic, œdematous, and more or less infiltrated. In the acute form the abscess is diffuse, but in long-standing cases the pus is encapsulated by a thick fibrous sac. The temporo-sphenoidal lobe and the cerebellum are the most frequent seats. Abscesses secondary to distant foci of suppuration are commonly multiple.

SYMPTOMS.—Abscesses following injury frequently run an acute course, and are characterized by high fever, rigors, headache, delirium, convulsions, vomiting, and coma.

In chronic cases the *general symptoms* are headache, irritability, mental impairment, vertigo, vomiting, irregular fever, stupor, pallor, and loss of flesh and strength. The *focal phenomena* vary with the location of the abscess. Involvement of the motor area may be attended with convulsions or paralysis in one limb; of the temporo-sphenoidal lobe, with deafness, and perhaps aphasia; of the occipital lobe, with hemianopia; of the cerebellum, with persistent vomiting and loss of coördination.

Diagnosis. *Cerebral Tumors.*—The history of traumatism or of some primary suppurating disease, such as otitis, bronchiectasis, empyema, ulcerative endocarditis; the presence of fever, and the absence of optic neuritis will indicate abscess.

Acute cases can rarely be distinguished from *suppurative meningitis.*

Prognosis.—Grave. When the focal symptoms indicate involvement of an accessible region like the motor area, temporo-sphenoidal lobe, or cerebellum, operative interference affords considerable hope of success.

Treatment.—When the abscess is located in one of the regions specified, the skull should be trephined and the pus evacuated. In other cases the application of wet cups to the neck, of ice-bags to the head, and the internal use of opium, bromide of potassium, or of chloral, may temporarily relieve the distress.

CRETINISM.

Definition.—A congenital affection, characterized by a lack of physical development, an abnormal condition of the thyroid gland, myxœdema, and idiocy or imbecility.

Etiology.—Beyond heredity no cause is known. The condition is endemic in the Alps and Pyrenees. Sporadic cases are also observed in other parts of the world.

Symptoms. *Endemic Cretinism.* — The stature is short (three or four feet); the head is large, flat antero-posteriorly and broad laterally; the eyes are wide apart; the nose is flat; the lips are thick; the tongue is large and may protrude from the mouth; the chest is narrow; the belly is prominent; the fingers are short; the genitalia are not developed; the subcutaneous tissues, especially at the root of the neck, are thickened from mucoid or fatty deposits; the thyroid gland is frequently enlarged; and the mental condition is that of idiocy.

Sporadic cases present the same features, but the thyroid, instead of being larger, is often atrophied.

Congenital conditions presenting to a limited extent the phenomena of cretinism, are termed *cretinoid.*

Treatment.—Encouraging results have followed the use of an extract of the thyroid gland.

SPINAL LEPTOMENINGITIS.

(Spinal Meningitis.)

DEFINITION.—An inflammation of the spinal pia mater not associated with infectious cerebro-spinal meningitis.

ETIOLOGY.—The infectious fevers, exposure to cold and wet, traumatism, and tuberculosis are the etiological factors.

PATHOLOGY. *Acute Form.*—The membranes are opaque, thickened, congested, and adherent. The fluid in the arachnoid space is increased. In very acute cases there is more or less purulent infiltration. The periphery of the cord is always involved.

Chronic Form.—The membranes are very thick and fused into one homogeneous fibrous mass.

SYMPTOMS. *Acute Form.*—The disease may begin with a chill, which is followed by moderate fever. There is intense pain in the back radiating along the course of the nerves. The back is exquisitely tender. The spinal muscles are rigid and contracted, sometimes so much so as to induce opisthotonos. The reflexes are increased. When the exudate is sufficient to make considerable pressure on the cord, paralytic phenomena develop, such as slight anæsthesia and partial paralysis of the extremities.

There are no cerebral symptoms unless the meninges of the brain are involved.

DIAGNOSIS. *Myelitis.*—In this affection there are marked paralysis and anæsthesia; involvement of the bladder and rectum; and the formation of bedsores

Rheumatism of the Muscles and Fibrous Tissues of the Back.—In this condition the joints are involved; the urine is highly acid; the pain does not follow the nerve-trunks; and the symptoms yield to the salicylates.

Tetanus.—The presence of a wound; the absence of fever; the early involvement of the jaw; and the absence of exquisite tenderness in the back will separate tetanus from meningitis.

PROGNOSIS.—Extremely grave. Recovery sometimes follows, but rarely without partial paralysis.

Chronic Leptomeningitis.—Pain in the back; stiffness of

muscles; hyperæsthesia and paræsthesia of the lower extremities, but rarely any anæsthesia; some loss of power; and increased reflexes.

TREATMENT.—An ice-bag, leeches, or cups may be applied to the spine. Sedatives like chloral, bromides, and morphine are usually required. Warm baths relieve the pain and lessen the rigidity. Ergot and iodide of potassium are recommended.

If the acute symptoms subside, iodide of potassium may be administered internally; blisters and mercurial inunctions may be applied to the spine, and massage and electricity to the affected muscles.

CHRONIC SPINAL PACHYMENINGITIS.

(Cervical Hypertrophic Pachymeningitis, Internal Pachymeningitis.)

DEFINITION.—A chronic inflammatory affection of the dura mater, characterized by severe pains in the head, shoulders, arms, and loins, followed by paresis, wasting, and anæsthesia.

ETIOLOGY.—Male sex, middle life, prolonged exposure to cold, lowered vitality, spinal concussion, alcoholism, and syphilis are predisposing factors. It may be secondary to inflammation of neighboring structures, such as the vertebræ in Pott's disease.

PATHOLOGY.—The membranes are thickened, opaque, and adherent; the vessels are dilated; and the spinal fluid is increased. In advanced cases the membranes are glued together and form a thick, homogeneous, fibrous mass. The cervical region is most commonly affected. The inflammation may extend to the cord and peripheral nerves.

SYMPTOMS.—Sharp pains radiating into the head, shoulders, arms, and loins, followed by loss of power, anæsthesia, wasting, and rigidity, particularly in the upper extremities. When the lower part of the cord is involved the same phenomena are observed in the legs, and the knee-jerk is increased. The duration of the disease is several years.

DIAGNOSIS.—*Chronic Poliomyelitis.*—The absence of pain and of anæsthesia will separate poliomyelitis from pachymeningitis.

Multiple Neuritis.—In this affection the pain is less marked in the back and more marked in the extremities, and the nerve-trunks are tender on pressure.

Spinal Irritation.—In this condition the spine is tender at certain spots, and there is no radiating pain, anæsthesia, or wasting.

Prognosis.—This depends on the extent and cause. When the involvement is slight or is due to syphilis, the prognosis should be guardedly favorable.

Treatment.—Absolute rest. Tonics are often indicated. Counter-irritation should be made along the cord by frequent blisters or the actual cautery. Morphine, antipyrin, or phenacetin may be required for the relief of pain. Iodide of potassium may be administered for its absorbent effect, and in syphilitic cases it should be given freely in conjunction with some mercurial.

ACUTE MYELITIS.

Definition.—An acute inflammation of the substance of the cord, characterized by marked disturbances of motion, sensation, and nutrition.

Varieties.—When only a transverse section is involved the condition is termed *transverse myelitis.* When a large vertical section is affected the disease is termed *diffuse myelitis.* When the gray matter is especially involved it is termed *central myelitis.*

Etiology.—Traumatism; exposure to cold, especially when the body is overheated; over-exertion; alcoholism; syphilis; or the infectious fevers may induce it. It is sometimes secondary to a hemorrhage or a morbid growth in the cord.

Pathology.—The membranes are usually injected and opaque. The substance of the cord is red and soft, and the line of demarcation between the gray and white matter is indistinct. In very acute cases the substance of the cord may flow out as a reddish, creamy fluid when the membranes are cut. Occasionally there are conspicuous hemorrhagic effusions (hæmatomyelitis).

Microscopic examination reveals destruction of the nerve-

elements, and in their place granular débris, fat-globules, red blood-corpuscles, and leucocytes.

Symptoms. *Acute Transverse Myelitis.*—Moderate fever (101°–103°), loss of appetite, coated tongue, and constipation, followed by pain in the back radiating into the limbs. With the pain there are often various forms of paræsthesia, as numbness, tingling, burning, etc. The muscles may be the seat of tremors or of convulsive seizures. There is frequently a sense of painful constriction—"girdle pain"—at the level of the disease. Paralysis soon develops, and may become more or less complete. The reflexes are generally increased when the lesion is above the lumbar enlargement; but if the latter is involved they are lost. The paralyzed muscles are flabby, but do not yield the reactions of degeneration; when, however, the reflexes are exaggerated the muscles often become rigid and contracted. At first there may be retention of urine and feces, but later there is frequently incontinence. Anæsthesia is more or less complete. Bedsores soon develop and add to the distress of the patient.

Death may result in a few days from extension upward and involvement of the respiratory muscles. In many cases life is prolonged for several weeks, death finally resulting from exhaustion induced by bedsores and cystitis. In rare cases there is a spontaneous arrest of the inflammation, and slow recovery follows, attended with partial paralysis.

Acute Central Myelitis —This resembles the former, but the trophic disturbances are much more marked and the duration is shorter. The disease is characterized by moderate fever and its associated phenomena, pain in the back, complete loss of power and of sensation, loss of reflexes, incontinence of urine and feces, rapid wasting of the muscles, and the early development of bedsores. The disease invariably proves fatal in from one to two weeks.

Diagnosis. *Acute Poliomyelitis.*—In this disease the bladder and rectum are not involved, and there are no sensory disturbances.

Landry's Disease, or Acute Ascending Paralysis.—In this affection trophic disturbances are absent; the bladder and rectum are not involved; and the loss of sensation is slight.

Multiple Neuritis.—The "girdle pain" is absent; the sphincters are not affected; bedsores are rare; and pain is more marked in the extremities than in the back.

Meningitis—The girdle pain is absent; the sphincters are not affected; the irritative phenomena are more marked than the paralytic.

Hemorrhage into the Cord.—The paralysis develops abruptly.

Prognosis.—Always extremely grave. Acute central myelitis is invariably fatal. In other cases recovery attended with partial paralysis occasionally follows.

Treatment.—If possible, the patient should be placed on a water-bed. To delay the formation of bedsores extreme cleanliness is essential. Both in retention and incontinence of urine the catheter should be used twice daily. In incontinence of urine and feces the discharges should be received on cotton-wool or oakum, which should be frequently renewed and the parts thoroughly cleansed. In the beginning an ice-bag or wet cups may be applied to the spine. Such remedies as ergot, belladonna, quinine, and mercury are frequently employed, but they seem to exert little influence. If recovery should follow, massage, electricity, and strychnine may be employed with the hope of restoring power to the paralyzed muscles.

CHRONIC MYELITIS.

Etiology.—Middle life, continued exposure to cold and wet, syphilis, alcoholism, gout, traumatism, and excesses are the predisposing factors. It may be secondary to Pott's disease.

Pathology.—The membranes are opaque and adherent. The whole cord has a grayish color; it is firmer than normal and somewhat contracted.

Microscopic examination reveals destruction of nerve-elements, and their replacement by an overgrowth of connective tissue.

Symptoms.—The disease begins gradually with numbness, tingling, or burning in the lower extremities, followed by a loss of power and sensation. The reflexes are generally exaggerated. The sphincters soon become involved. The muscles do

not waste until the disease is far advanced. As in other organic affections of the cord, there is often a sense of constriction, or "girdle pain," at the level of the disease. The disease progresses very slowly, the duration being from six months to ten years.

Diagnosis.—The diagnosis rests on the gradual development of symptoms indicating a general involvement of the cord.

Treatment.—The patient should be put at rest; tonics are often indicated; counter-irritation to the spine by repeated blisters or applications of the actual cautery, often yields good results. The frequent use of tepid baths is also beneficial. The special remedies which have been recommended are arsenic, strychnine, phosphorus, nitrate of silver, mercury, and iodide of potassium. When there is a suspicion of syphilis the last two remedies should be given a thorough trial.

SCLEROSIS OF THE SPINAL CORD.

Definition.—A degenerative affection of the spinal cord, characterized anatomically by an atrophy of the nerve-elements and an overgrowth of connective tissue.

Etiology.—Middle life, male sex, syphilis, alcoholism, mineral poisoning, excesses, and continued exposure to cold and wet are the usual causes.

Locomotor Ataxia.

(Locomotor Ataxy, Tabes Dorsalis, Posterior Sclerosis.)

Definition.—A degenerative affection of the lower sensory neurons, involving the spinal ganglia, posterior roots, and posterior columns of the cord, and characterized by incoordination, loss of deep reflexes, disturbances of nutrition and of sensation, and various ocular phenomena.

Pathology.—The membranes over the posterior columns are often opaque and adherent. The posterior columns have a grayish color, and are firm and shrunken.

Microscopic examination reveals atrophy of the nerve-fibres and an overgrowth of connective tissue. Degenerative

changes are also observed in the posterior roots of their ganglia. Not infrequently the process involves the basal ganglia of the brain and cranial nerves.

Symptoms. *Motor Phenomena.* — One of the earliest symptoms is loss of coördination. This is first manifested by unsteadiness when the patient walks in the dark. When he stands erect, with the eyes closed and feet together, he staggers and tends to fall (Romberg's symptom). When the arms are affected there is inability to perform work requiring delicate coördination, such as writing and piano-playing. This loss of coördination in the upper extremities becomes conspicious when the patient, while his eyes are closed, attempts to touch the tip of his nose.

The gait is characteristic; in walking he raises his feet high, throws them forwards, and brings them down forcibly in such a way that the whole sole strikes the floor at once. Although the patient may be unable to walk or to use his hands with precision, there is no actual loss of power.

Sensory Phenomena.—Pain is rarely absent; it is sharp and lancinating in character, and appears in paroxysms. It usually involves the extremities, but sometimes it attacks the stomach and is accompanied with obstinate vomiting. The term *gastric crisis* is applied to this phenomenon.

Crises may occur in other organs, notably the larynx, where they are manifested by intense dyspnœa and stridulous breathing. Various forms of paræsthesia are observed, such as tingling, numbness, "pins and needles," and the like. Irregular areas of anæsthesia are frequently distributed over the body.

Reflexes.—The patellar reflex is lost very early in the disease. The pupil fails to respond to light while it still accommodates for distance (Argyll-Robertson pupil).

Eye Phenomena.—The most important are diplopia, contracted pupils, dimness of vision from optic atrophy, and paresis of the ocular muscles.

Trophic Phenomena.—The most curious are the so-called arthropathies, which consist of enlargement of the joints, associated with serous effusions, atrophy of the heads of the bone, erosion of the cartilages, and calcification of the ligaments. These articular changes sometimes lead to luxations.

Perforating ulcer of the foot is sometimes observed.

Other symptoms sometimes observed are: loss of sexua power, paralysis of the sphincters, epileptiform seizures, and dementia.

Diseases with which locomotor ataxia may be associated.—Spastic paraplegia, multiple neuritis, paretic dementia, and chronic poliomyelitis.

Diagnosis. *Multiple Neuritis.*—In this affection the peripheral nerves are tender; the muscles may yield the reactions of degeneration; the pain is not lancinating like that of ataxia; and the Argyll-Robertson pupil is absent.

Tumor of the Cerebellum.—In this condition the reflexes are not abolished, lightning pains are absent, and instead there are persistent vomiting, headache, and optic neuritis.

Gastralgia.—A gastric crisis may be mistaken for gastralgia, but the associated phenomena of locomotor ataxia will prevent an error in diagnosis.

Prognosis.—Generally unfavorable, although arrest and even improvement are not infrequent. The duration is indefinite.

Treatment.—The patient should be placed under the best hygienic conditions. Rest is desirable. In the early stage a prolonged voyage may produce excellent results. The diet must be nutritious, but easily assimilable. Excesses of all kinds must be rigidly prohibited. Tonics are frequently indicated. When there is a suspicion of syphilis, iodide of potassium should be given in full doses. In other cases iodide of potassium in small doses, mercury, and arsenic, are the most reliable remedies. The following pill may prove useful:—

℞ Sodii arsenat.,
Zinc. phosphid., āā gr. ij;
Hydrarg. iodid. rub., gr. j.—M.
Ft. in pil. No. xxx.
Sig.—One, three times daily after meals.

Fränkel's method of reteaching the lost power of coördination by graduated exercises is of decided value.

The Pains.—When very intense, morphine will be required; in other cases antipyrin, phenacetin, and cannabis indica are sometimes efficient.

℞ Antipyrin, ℥j ;
Syr. zingiber., f℥j ;
Aquæ q. s. ad f℥iv.—M. (Germain See.)

Sig.—A teaspoonful every one to four hours for three to six doses.

The *laryngeal crises* may be relieved by the inhalation of chloroform or amyl nitrite.

Primary Spastic Paraplegia.

(Lateral Sclerosis, Antero-lateral Sclerosis.)

Definition.—A nervous affection probably dependent upon sclerosis of the lateral columns, and characterized by loss of power, increased reflexes, and a spastic condition of the muscles.

Pathology.—There is probably a sclerosis of the lateral columns of the cord.

Symptoms.—Loss of power is generally the first symptom. This begins in the lower extremities and increases very slowly. The knee-jerk is exaggerated, and in most cases ankle-clonus can be elicited. When put in use the muscles become stiff, or spastic, and when the disease is fully developed the gait is peculiar. In walking the knees are drawn together, the legs drag behind, and the toes catch the ground.

The muscles do not waste, but rather tend to become hypertrophied from continued reflex stimulation. The sphincters are ultimately affected. Sensation is generally undisturbed, but subjective phenomena like numbness and tingling may be observed. The upper extremities are not often involved, but finally loss of power and rigidity may develop in them also.

Prognosis.—Unfavorable. In rare instances the disease is arrested.

The duration is indefinite.

Treatment.—The general treatment is the same as in locomotor ataxia. For the spasmodic condition of the muscles, rubbing, warm baths, and the following remedies are recommended: bromide of potassium, calabar bean, and belladonna.

Amyotrophic Lateral Sclerosis.

DEFINITION.—A nervous affection characterized anatomically by a degeneration of the lateral columns and adjacent gray matter, and manifested clinically by loss of power, wasting, and a spastic condition of the muscles.

PATHOLOGY.—The disease apparently depends upon a sclerosis involving mainly the anterior horns of the gray matter and the antero-lateral columns.

SYMPTOMS.—Loss of power and wasting, usually beginning in the small muscles of the hand, and gradually spreading over the entire body. The reflexes are exaggerated. When the muscles are put into use, they become more or less rigid, or spastic. The degenerative process extends upwards until it involves the medulla, when symptoms of bulbar palsy appear.

DIAGNOSIS.—The muscular rigidity and exaggerated reflexes will distinguish it from pure *progressive muscular atrophy.*

PROGNOSIS.—Unfavorable.

TREATMENT.—Such remedies as arsenic and iodide of potassium are recommended, but they usually prove useless. The spastic condition is improved by massage.

Ataxic Paraplegia.

DEFINITION.—A sclerotic affection of the posterior and lateral columns manifesting symptoms of both locomotor ataxia and spastic paraplegia.

SYMPTOMS.—It resembles spastic paraplegia in the loss of power, spastic condition of the muscles, increased reflexes, and absence of sensory disturbances; and locomotor ataxia in the distinct loss of coördination.

Disseminated Cerebro-spinal Sclerosis.

(Multiple Sclerosis, Insular Sclerosis.)

DEFINITION.—A chronic nervous disease characterized anatomically by patches of sclerosis of varying size scattered through the brain and cord.

ETIOLOGY.—The causes which lead to other scleroses of the cord may induce this disease; the infectious fevers, however,

are assigned a prominent place in its etiology. It is more commonly observed in younger people than is locomotor ataxia or lateral sclerosis.

PATHOLGY.—Areas of firm, gray, sclerotic tissue, of various sizes and shapes, are found through the brain and cord.

SYMPTOMS.—The spinal symptoms may resemble either locomotor ataxia or lateral sclerosis, according as the posterior or lateral columns are chiefly affected. The characteristic symptoms are loss of power, usually most marked in the legs; increased reflexes; vague pains; a coarse tremor developed on movement (volitional tumor); a slow, hesitating, "scanning" speech; nystagmus—tremor of the eyeballs; and mental impairment. Sensory and trophic disturbances are generally absent.

DIAGNOSIS.—Disseminated sclerosis may be mistaken for *paralysis agitans*, but the latter disease develops in late life; the tremor is fine, rarely involves the head, and is not made worse by use of the muscles; and nystagmus is absent.

PROGNOSIS.—Unfavorable. The duration is indefinite, and long remissions with improvement of the symptoms are not uncommon.

TREATMENT.—The general treatment is the same as that for posterior sclerosis. Bromides, hyoscine, hyoscyamine, and belladonna have been recommended for the tremors.

Hereditary Ataxia.

(Friedreich's Disease.)

DEFINITION.—A sclerotic affection of the spinal cord, occurring in several children of the same family, and characterized by symptoms resembling locomotor ataxia.

ETIOLOGY.—The greatest number of cases develop between the second and fifteenth years. Some can be traced to hereditary influence; in others a cause cannot be ascertained.

PATHOLOGY—Sclerosis of the posterior and lateral columns of the cord.

SYMPTOMS.—Loss of coördination in the arm and legs, nystagmus, irregular jerking movements of the hands, loss of

reflexes, a scanning speech, spinal curvature, equino-varus (heel raised and the sole turned in).

It differs from locomotor ataxia in the absence of sharp pains, of anæsthesia, and of the Argyll-Robertson pupil, and in the occurrence of irregular movements of the hands, nystagmus, scanning speech, and equino-varus.

PROGNOSIS.—Unfavorable. The duration is many years.

SYRINGO-MYELIA.

DEFINITION.—A chronic affection of the spinal cord characterized anatomically by the formation of a cavity in its substance, and clinically by atrophy of certain muscles, peculiar disturbances of sensation, and various trophic disorders.

ETIOLOGY.—It is much more common in males than females. Eighty per cent. of the cases occur between the ages of ten and forty years. Traumatism or one of the infectious fevers may excite it.

PATHOLOGY.—The disease begins as an overgrowth of the embryonic neuroglia. The cavity-formation is a secondary process, and is brought about by degeneration of the gliomatous tissue, or possibly in some instances by hemorrhage. The cervical and upper dorsal regions are the usual seats of the lesion. The cavity lies in the gray matter, and may be in the position of the central canal or somewhat posterior to it. Secondary degenerations are frequently observed in the anterior or posterior horns or in the anterior or posterior columns.

SYMPTOMS.—The disease usually attacks the upper extremities, the chief symptoms being: Wasting of the muscles; fibrillary tremors; loss of painful and thermic sensations, while tactile sensation is preserved or but slightly affected (*dissociation symptom*); lateral spinal curvature; and various trophic disturbances, such as arthropathies, fissures, ulcers, and gangrene. Such eye symptoms as nystagmus, inequality of pupils and narrowing of the visual fields are frequently observed. In many instances symptoms of lateral sclerosis, posterior sclerosis, or bulbar disease are superadded.

The distinctive features of *Morvan's disease* (probably a form of syringo-myelia) are tactile anæsthesia and painless felons.

DIAGNOSIS.—*Cervical pachymeningitis* is more painful, and the anæsthesia includes tactile sensation. In *progressive muscular atrophy* and *amyotrophic lateral sclerosis* sensory symptoms are wanting. *Leprosy* may be recognized by loss of tactile sensation, discoloration of skin, nodular swellings, and presence of bacilli in the secretions of the nose and eyes and in the serum of blisters.

PROGNOSIS.—Unfavorable. The duration is from five to twenty years.

TREATMENT.—This is necessarily symptomatic.

ACUTE ANTERIOR POLIOMYELITIS.

(Infantile Paralysis, Atrophic Spinal Paralysis.)

DEFINITION. — An acute disease, occurring almost exclusively in young children, characterized anatomically by a destruction of the ganglion-cells in the anterior gray horns of the cord, and manifested clinically by abrupt paralysis and rapid wasting of certain muscles.

ETIOLOGY. — The greatest number of cases occur within the first three years, and the disease is far more common in summer than in winter. The sudden onset, the absence of any known exciting cause, and the fact that it has occurred epidemically suggest an infectious origin.

PATHOLOGY. — The sudden onset and wide-spread initial paralysis are probably due to intense congestion, and the permanent paralysis and wasting to destruction of the ganglion-cells in the anterior gray horns. Microscopic examination in recent cases reveals ecchymoses, destruction of ganglion-cells, and infiltration of leucocytes.

Examination long after the development of the paralysis reveals an absence or atrophy of the large multipolar cells in the gray horns, and in their stead an overgrowth of connective tissue. The anterior nerve-roots and muscles also reveal degenerative changes.

SYMPTOMS.—Generally the onset is abrupt; often the child is put to bed in apparent health and in the morning is found paralyzed in one or more limbs. In some cases febrile symptoms precede the attack, and more rarely the disease is ushered in with a chill, a convulsion, or delirium.

The paralysis at first may be quite extensive, but more commonly it confines itself to certain groups of muscles in the upper or lower extremities. The latter are especially prone to suffer; the affected muscles are relaxed, and the surface is cold and often cyanosed. The paralysis is peculiar in its irregular distribution and in its tendency to improve spontaneously up to a certain limit. There are no sensory disturbances, no involvement of the bladder and rectum, and no tendency to bedsores. The muscles which are permanently affected rapidly waste and ultimately yield the reactions of degeneration. From contractures of the atrophied muscles, and contraction of their healthy antagonists, various deformities develop.

Diagnosis.—The abrupt onset will distinguish it from both *idiopathic muscular atrophy* and *progressive muscular atrophy*. The absence of sensory disturbances, bedsores, and paralysis of the bladder and rectum will separate it from *myelitis*. The presence of cerebral symptoms, of choreiform or athetoid movements in the affected members, and the absence of reactions of degeneration and of early wasting will separate *cerebral paralysis of childhood* from acute poliomyelitis.

Prognosis.—Unless the initial symptoms are very severe, the prognosis, as regards life, is good. In all cases some of the paralysis disappears. Occasionally the improvement is so great that the usefulness of the member is not impaired; but far more frequently the residual paralysis is sufficient to cause considerable deformity and disability.

Treatment.—During the acute stage the child should be confined to bed. To relieve the congestion, dry cups may be applied to the spine and ergot may be given internally. The affected members should be wrapped in flannel.

After the lapse of two or three weeks electrical treatment should be instituted; the faradic current may be employed when it induces contraction of the affected muscles, but when it excites no response the galvanic current must be substituted. Massage is a very valuable adjunct to the electrical treatment. Internally strychnine (gr. $\frac{1}{100}$ to a child of two years) gradually increased is a useful muscular stimulant. Massage and the adjustment of mechanical appliances will be required to combat deformity from contractures.

PROGRESSIVE MUSCULAR ATROPHY.

(Chronic Spinal Muscular Atrophy, Chronic Poliomyelitis.)

DEFINITION.—A chronic nervous affection, characterized anatomically by degeneration of the ganglion-cells of the gray matter in the cord, and manifested clinically by loss of power and atrophy of corresponding muscles.

ETIOLOGY.—Male sex, middle life, and hereditary tendency are the predisposing causes. It sometimes follows prolonged emotional excitement, exposure to cold, traumatism, and syphilis.

PATHOLOGY.—Microscopic examination of the gray matter of the cord reveals atrophy or complete absence of the large multipolar cells in the anterior cornua, and an overgrowth of connective tissue. The anterior root-fibres are also the seat of degenerative changes. In some cases the lateral columns are likewise sclerosed (amyotrophic lateral sclerosis).

Examination of the affected muscles reveals atrophy of the fibres, fatty degeneration, an overgrowth of connective tissue, and an absence of transverse striation, and instead, longitudinal striation.

SYMPTOMS.—Not infrequently prodromal symptoms are noted in the parts to be affected, such as pain, coldness, or numbness. Soon, loss of power and wasting begin in the small muscles of the hand, namely, the thenar and interossei muscles. Although one hand is usually affected before the other, the disease tends to become symmetrical. Next to the hands the muscles of the shoulders and arms slowly waste, rendering the bony prominences marked; and so the disease advances little by little until the patient is reduced to a mere skeleton. The hands assume a characteristic appearance: from atrophy of the interossei and contraction of the long extensor and flexor muscles they become "claw-like." The wasted muscles are frequently the seat of fibillary tremors. The response to the galvanic and faradic currents is diminished, but the reactions of degeneration do not develop until the disease is far advanced. Although the patient may complain of coldness

and numbness, sensation is not impaired. The legs are not involved until late, and often escape entirely.

The wasting progresses very slowly, and death may result from some intercurrent disease; if such is not the case, extension to the medulla leads to symptoms of *bulbar palsy*, such as indistinct articulation, inability to pucker the lips, difficult swallowing, and embarrassed respiration.

COMPLICATIONS.—It may be associated with *lateral sclerosis*, when it is termed *amyotrophic lateral sclerosis*. It may lead to *bulbar palsy*.

DIAGNOSIS. *Primary Muscular Atrophy.*—This disease develops in earlier life, rarely begins in the hand, and the hereditary tendency is more marked than in poliomyelitis.

PROGNOSIS.—Always unfavorable. The duration is indefinite.

TREATMENT.—Good hygiene. Nutritious food. Tonics. Gowers claims good results from the hypodermic injection of nitrate of strychnine (gr. $\frac{1}{100}$ increased to gr. $\frac{1}{40}$) once daily. Massage and electr[illegible] results.

BULBAR PARALYSIS.

(Glosso-labio-laryngeal Paralysis.)

DEFINITION.—Paralysis of the lips, tongue, pharynx, and larynx from destruction of the ganglionic cells of the medulla oblongata.

ETIOLOGY.—An acute form is observed which results either from hemorrhage or from an acute poliomyelitis of the medulla. The chronic form, or progressive bulbar palsy, may result from chronic poliomyelitis involving primarily the medulla, or from the extension of the degenerative process in paretic dementia, amyotrophic lateral sclerosis, progressive muscular atrophy, or acute ascending paralysis (Landry's disease).

SYMPTOMS.—Impairment of speech; inability to protrude the tongue; dribbling of saliva; difficult swallowing; choking spells from the entrance of food or mucus into the larynx; partial suppression of the voice and measured speaking; fibrillary tremors of the lips and tongue; loss of reflex action;

atrophy of the lips, tongue, and pharynx; and, finally, difficult respiration and disturbed cardiac rhythm.

PROGNOSIS.—Unfavorable. The acute variety is speedily fatal; the chronic form may last several years. Death may result from exhaustion, cardiac failure, or aspiration-pneumonia.

TREATMENT.—Electricity, strychnine, and the use of a stomach-tube when swallowing becomes difficult.

ACUTE ASCENDING PARALYSIS.

(Landry's Disease.)

DEFINITION.—An acute disease of rare occurrence, characterized by motor paralysis, beginning in the feet and rapidly spreading until it involves the muscles of respiration and deglutition.

ETIOLOGY.—The causes are unknown. It is usually observed in young male adults. The abr··· ·t, acute course, and absence of known cause and of · , lesions have suggested an infectious origin.

PATHOLOGY.—No demonstrable lesions have been discovered.

SYMPTOMS.—Febrile symptoms usually usher in the attack. The paralysis begins in the legs and involves successively the trunk, upper extremities, and muscles of respiration and deglutition. The reflexes are abolished. The sphincters are retentive; sensation is usually normal, but there may be paræsthesia or some anæsthesia; the muscles are relaxed, but do not waste or yield the reactions of degeneration. In some instances the spleen and lymphatic glands are swollen.

DIAGNOSIS. *Acute Myelitis.*—Anæsthesia, wasting, reactions of degeneration, and early involvement of the sphincters will serve to distinguish myelitis from acute ascending paralysis.

Multiple neuritis will be separated from Landry's disease by the marked sensory disturbances in the former.

PROGNOSIS.—Unfavorable. The vast majority of cases terminate fatally in the course of a few days. Occasionally there is a spontaneous arrest, and a gradual restoration to health.

TREATMENT.—Cups to the spine and electricity to the affected muscles have been employed with indifferent results.

CAISSON DISEASE.

(Divers' Paralysis.)

DEFINITION.—A condition observed in divers and others subjected to increased atmospheric pressure, and characterized by motor and sensory paralysis and other nervous symptoms.

ETIOLOGY.—A pressure of more than two atmospheres is required to produce the paralysis, and the time elapsing before its appearance lessens as the pressure increases.

PATHOLOGY.—The symptoms have been ascribed by some to the liberation in the cord of gases which have been absorbed by the blood during exposure to the high pressure; by others, to stasis of blood and œdema. The cord is found congested and sometimes the seat of hemorrhages.

SYMPTOMS.—The condition may manifest itself immediately on reaching the surface or after the lapse of several hours. The most important phenomena are pains in the joints followed by motor and sensory paralysis in the lower extremities. The bladder and rectum are sometimes involved. Occasionally the paralysis takes the form of a hemiplegia instead of a paraplegia. Gastralgia and vomiting are common symptoms. In severe cases coma develops and death follows in a few hours. Generally, however, the symptoms gradually subside, and the power is fully restored in the course of a few days or a few weeks.

TREATMENT.—As a preventive measure the transition from high to low pressure should be accomplished gradually. Marked cases should be treated as acute myelitis.

IDIOPATHIC MUSCULAR ATROPHY.

(Muscular Dystrophy, Myopathic Atrophy.)

DEFINITION.—An atrophic condition of the muscles developing in early life and not dependent upon any lesion in the nervous system.

ETIOLOGY.—The disease usually manifests itself before puberty. It is more common in males than in females. It

is frequently transmitted from generation to generation, and several members of the same family may be similarly affected.

Pathology.—No lesion in the cord or nerves is observed. Gowers regards the disease as of developmental origin. Microscopic examination of the muscles reveals atrophy of their fibres and an unnatural amount of fat and connective tissue. When the latter elements are considerably increased, a pseudo-hypertrophy results (pseudo-muscular hypertrophy).

Symptoms.—The muscles, especially those of the face, shoulders, thighs, buttocks, and calves, lose power and waste. Fibrillary twitchings are rarely noted. The reactions of degeneration are absent. In *Erb's juvenile* type the atrophy begins in the shoulder; in the *Landouzy-Déjérine* type, in the face.

Diagnosis. *Chronic Poliomyelitis.*—This disease develops later in life without marked hereditary tendency, and nearly always begins in the small muscles of the hands—parts which are rarely affected in idiopathic atrophy.

Multiple Neuritis.—Pain, anæsthesia, paræsthesia, the history, and the distribution of the palsy will suggest neuritis.

Prognosis.—Unfavorable. The disease is incurable, but of slow progress.

PSEUDO-HYPERTROPHIC PARALYSIS.

(Pseudo-muscular Hypertrophy, Lipomatous Muscular Atrophy.)

Definition.—A disease of childhood, characterized by paralysis depending upon degeneration of the muscles, which, however, become enlarged from a deposition of fat and connective tissue.

Etiology.—Male sex, childhood, and an hereditary tendency are the only known predisposing causes. Several cases have frequently been observed in the same family.

Pathology.—The disease is allied to idiopathic muscular atrophy, with which it is frequently associated. Since no lesions are observed in the cord or peripheral nerves it is to be regarded as a primary affection of the muscles. Microscopic examination reveals an excessive amount of fat and connective

tissue between the muscle-fibres, the latter being atrophied and more or less degenerated.

Symptoms.—The first symptom to attract attention is weakness of the muscles; the child is awkward, stumbles, and in walking seeks support. As the paralysis increases, the muscles, particularly those of the calf, thigh, buttock, and back, enlarge. The upper extremities are less frequently affected. When the child assumes the erect posture the feet are wide apart, the belly protrudes, and the spinal column shows a marked curvature with the convexity forward. The manner of rising from the recumbent position is characteristic: He straightens himself either by grasping the knees, or by resting the hands on the floor in front of him, extending the legs, and pushing the body backwards. The gait is waddling in character.

Although the response of the muscles to electrical currents is less pronounced, the reactions of degeneration are not present. The knee-jerk is lessened or abolished. There are no mental or sensory disturbances.

In the course of a few years, the paralysis becomes so marked that the patient is unable to leave his bed; the enlargement of the muscles is followed by atrophy; and finally death results from some intercurrent disease, or inflammation of the lungs induced by the weakened respiratory power.

Prognosis.—Absolutely unfavorable.

Treatment.—Remedies generally prove useless. Graduated exercise, massage, electricity, and hypodermics of strychnine may be employed with the hope of staying the progress of the disease.

NEURALGIA.

Definition.—Paroxysmal pain radiating along the course of a nerve-trunk.

Etiology.—Heredity, female sex, nervous temperament, excesses, overwork, and nervous exhaustion are general predisposing factors. It is frequently an expression of anæmia. It may result from the action of some toxic agent in the blood; thus it is common in malaria, rheumatism, gout, syphilis, and

chronic lead-poisoning. It may be caused by reflex irritation; thus a trifacial neuralgia may depend on caries of the teeth or eye-strain. In some cases neuralgia results from organic disease of the nerve-centre; thus obstinate trifacial neuralgia may be dependent upon some degeneration or tumor of the Gasserian ganglion.

Exposure to cold and wet frequently acts as an exciting cause in susceptible people.

Pathology.—The pathological condition upon which neuralgia depends is unknown. In many cases, no doubt, it is a manifestation of neuritis.

Symptoms.—Certain prodromes frequently give warning of an approaching attack; these are chilliness, depression of spirits, and perhaps tingling in the part to be affected. The chief symptom is intense pain, which is usually of a sharp, stabbing character. The area supplied by the affected nerve is generally hyperæsthetic, and palpation detects spots of exquisite tenderness where the nerve makes its exit through a bony canal or fibrous sheath; the latter have been termed Valliex's points. In some cases the pain is attended with severe clonic or tonic spasms of the muscles. Inspection of the part usually reveals negative results, but occasionally distinct swelling or an outbreak of herpes is observed.

The attack lasts from a few minutes to many hours, and its subsidence may be marked by the passage of a large amount of pale urine. The interval between the paroxysms varies in different cases; it is frequently several weeks or months. It is noteworthy that the attacks often recur at regular intervals.

Trifacial Neuralgia (*Tic Douloureux*, *Prosopalgia.*)—In this variety the pain involves one or more branches of the trifacial nerve. The tender points correspond to the supra-orbital, infra-orbital, and mental foramina. Violent spasms of the muscles are frequently observed. In long-standing cases the hair on the affected side may become coarse and bleached. Trifacial neuralgia is frequently reflex, being dependent upon caries of the teeth, eye-strain, nasal disease, or some distant centre of irritation.

Intercostal Neuralgia.—In this variety the pain follows the course of the intercostal nerves. It is frequently associated

with an eruption of herpes zoster. Spots of tenderness may be detected near the vertebral columns, in the middle of the nerve, and near the sternum. The frequent dependence of intercostal neuralgia upon spinal caries or thoracic aneurism must not be forgotten.

Occipital neuralgia involves the upper cervical nerves. A spot of tenderness may be discovered midway between the mastoid process and the upper cervical vertebræ. This form of neuralgia may be an expression of spinal caries.

Sciatica has been described elsewhere.

Diagnosis. *Neuritis.*—The continuous pain, the tenderness along the entire nerve, the presence of paræsthesia, anæsthesia, paresis, and wasting will serve to distinguish neuritis from neuralgia.

The lightning-pains of locomotor ataxia must not be mistaken for neuralgia. The abolished patellar reflex, the loss of coördination, and the Argyll-Robertson pupil in the former will indicate the diagnosis.

Prognosis.—For the attack the prognosis is good; for permanent cure, it must be guarded. When the cause can be removed the prognosis is favorable.

Treatment. *The Attack.*—The patient should be kept in a quiet, cool, well-ventilated room. Local applications are useful; hot cloths, stimulating liniments, an ointment of aconitine, a small blister, or a hypodermic injection of cocaine, chloroform, or morphine and atropine may be employed. One of the following applications will prove serviceable:—

℞ Aconitinæ, gr. iv;
Veratrinæ, gr. xv;
Glycerini, ʒij;
Cerati, ʒvj.—M. (Da Costa.)

Sig.—To be rubbed over the parts. Do not apply to any abrasion of the skin.

Or—

℞ Chloral. hydrat.,
Pulv. camphor., āā ʒss.—M.

Sig.—Apply with a camel's hair brush.

Internally, antipyrin, phenacetin, cannabis indica, bromide of potassium, butyl chloral, and exalgine are efficient remedies.

Morphia is sometimes required, but the danger of inducing the habit should always be borne in mind.

The Interval.—Careful search should be made for an exciting cause, which, if found, must be removed. The teeth, eyes, nose, gastro-intestinal tract, urine, and blood should be carefully examined.

In anæmia, iron and arsenic are indicated; in syphilis, iodide of potassium; in rheumatism, salicylate of sodium or iodide of potassium; in malaria, quinine and arsenic; in gout colchicum and lithium; in lead-poisoning, iodide of potassium.

Tonics like iron, quinine, strychnine, cod-liver oil, and phosphorus are frequently indicated. Among the special remedies may be mentioned arsenic, velerian, hyoscyamus, aconitia, gelsemium, cannabis indica, oxide of zinc, nitro-glycerin, and asafoetida. The following pill, devised by Dr. S. D. Gross, is often very useful :—

℞ Quinin. sulph., ʒj
Morphin. sulph.,
Acid. arsenosi, āā gr. iss;
Ext. aconiti, gr. xv;
Strychnin. sulph., gr. j.—M.
Ft. in pil. No. xxx.
Sig.—One, thrice daily.

Local treatment in the interval may accomplish much. Electricity, acupuncture, or repeated blisters may be employed. In obstinate cases surgical interference may be required to secure relief. Three operations have been performed: Nerve-stretching; neurotomy, or section of the nerve; and neurectomy, or removal of a portion of the nerve.

MIGRAINE.

(Hemicrania, Megrim, Sick-headache.)

Definition.—Paroxysmal circumscribed headache associated with visual, vaso-motor, and gastric disturbances.

Etiology.—It is frequently hereditary. It is more common in women than in men. It usually develops in early life. Anæmia, gastric disturbances, gout, eye-strain, menstrual disorders, overwork, and prolonged excitement predispose to it.

PATHOLOGY.—Unknown. There is a tendency to regard it as an indication of hereditary degeneration.

SYMPTOMS.—The attack is often preceded by malaise, restlessness, and perverted vision. The pain is sharp and stabbing and frequently limited to the temporo-frontal region of one side. The surface is extremely hyperæsthetic, but the tender spots noted in trifacial neuralgia are absent. The patient is very sensitive to light and sound, and during the attack usually confines herself to a darkened room. Nausea and vomiting are frequently present. In some cases the temporal artery is contracted, the face is pale, and the pupil large; in others the artery is dilated, the face is flushed, and the pupil small. The duration of the attacks varies from a few hours to several days. In the intervals, which are often of definite duration, the patient may be quite well.

Less frequent symptoms are vertigo, hallucinations of sight, cramps of the facial muscles, tingling or numbness in one hand, partial aphasia, and paresis of the ocular muscles.

PROGNOSIS.—Perfect cure is rare, but the severity and frequency of the seizures may be considerably lessened by treatment.

TREATMENT. *The Attack.*—Rest in a darkened, quiet, and well-ventilated room; antipyrin, caffeine, bromide of potassium, salol, and morphine with atropine are useful remedies.

℞ Antipyrin, ʒj;
Syr. aurant. cort., fʒj;
Aquæ, q. s. ad fʒiij.—M.
Sig.—A tablespoonful every two hours.

Or—

℞ Caffein. citrat., gr. xij;
Phenacetin, gr. xviij;
Sodii bromid., ʒj.—M.
Ft. in chart. No. vi.
Sig.—One powder every hour.

Or—

℞ Salol, ʒj;
Caffein. citrat.,
Phenacetin, āā gr. xviij.—M.
Ft. in chart. No. vi.
Sig.—One every two hours.

The Interval.—Careful search should be made for some exciting cause, and this removed, when possible. The habits of the patient must be regulated. Overwork and the use of alcohol, strong tea and coffee must be interdicted. Systematic exercise and frequent bathing followed by friction are valuable adjuncts. The diet must be adapted to the condition of the stomach and the needs of the system. Internally, arsenic, iodide of potassium, bromide of potassium, valerianate of zinc, and cannabis indica are the most reliable remedies. Cannabis indica is often very efficient, and a quarter to half a grain of the extract may be given for a prolonged period. Little recommends :—

℞ Sodii arsenat., gr. ij ;
Ext. cannabis indicæ, gr. iv ;
Ext. belladonnæ, gr. viij.—M.
Ft. in pil. No. xxiv.
Sig.—One, twice daily.

HEADACHE.

(Cephalalgia.)

Definition.—Pain in the head generally resulting from a disturbance of the cerebral circulation, a perverted condition of the blood, reflex irritation, or pressure on the brain by inflammatory exudate, depressed bone, or a tumor.

Organic Headache.—This form is observed in meningitis, cerebral tumor, abscess, softening, etc., and may be recognized by its persistence and by the associated evidences of organic cerebral disease, such as optic neuritis, mental aberration, paralysis, especially of the facial muscles, and vomiting arising independently of other gastric symptoms.

Under this head is included the headache of *syphilis*, which may be diagnosed by the history ; by the other evidences of syphilis ; by its frequent association with somnolence ; and by the effect of iodide of potassium.

Headache of Cerebral Hyperæmia.—*Active cerebral congestion* usually results from prolonged mental work, fever, or exposure to the sun. Toxic and reflex headaches are often directly due to active cerebral congestion, but these will be discussed later.

Passive cerebral congestion may result from obstruction to the return of blood from the brain, as by a tumor of the neck, or cardiac disease. It is also common in elderly people from a relaxed condition of the vessels.

In cerebral congestion the headache is of a throbbing or bursting character; the head is hot; the face flushed; the eye-ground injected; and the distress is increased by lowering the head.

The exciting cause must be determined by the history and by a careful examination of the various organs, especially the heart.

Headache of Cerebral Anæmia.—This is frequently dependent upon general anæmia. It is also common in neurasthenia resulting from overwork, prolonged emotional excitement, excesses, etc. More rarely it is dependent upon aortic stenosis.

In cerebral anæmia the pain is frequently vertical; it is not throbbing, but it is described as a sensation of weight or gnawing; the extremities are cold; the face and eye-grounds are pale; the mind is depressed; fainting spells are often present; lowering the head and the inhalation of nitrite of amyl relieve the pain.

Reflex Headache.—Headache is often due to *eye-strain* resulting from refraction errors, and in obstinate cases a careful examination of the eyes should always be made. Headache of this origin is frequently a browache, and may be associated with restlessness, vomiting, and insomnia. It is induced or aggravated by prolonged use of the eyes.

Ovarian or uterine diseases often produce a reflex headache. It is usually located at the vertex, and is relieved by pressure of the hand.

Gastric irritation is responsible for many headaches; the latter are invariably relieved by vomiting, and are usually associated with other evidences of stomachic disorder.

Nasal catarrh may induce persistent headache, which is generally confined to the forehead, temples, or vertex, and is aggravated by exacerbations of the catarrah. The pain is often associated with tenderness of the inner wall of the orbit,

and is increased by irritating the nasal mucous membrane with a probe.

Toxæmic Headache.—A persistent headache often results from Bright's disease, and is *urœmic* in origin. It may be recognized by the high arterial tension and by the albumin and casts in the urine. A urinary analysis should be made in all cases of persistent headache.

Gout or lithœmia produces an intractable headache which is associated with vertigo, great irritability of temper, and a "brick-dust" deposit in the urine.

Chronic malarial poisoning may manifest itself in a headache which is usually confined to the supraorbital region. It is apt to recur at regular intervals, is often associated with tenderness over the supraorbital nerve, and is only relieved by large doses of quinine.

A headache of *rheumatic* origin sometimes develops in those subject to rheumatism. It is frequently excited by exposure or a sudden change of temperature. It usually affects the aponeurosis of the occipito-frontalis and temporal muscles, is increased by wrinkling the forehead and forcibly moving the jaws, and is associated with tenderness of the scalp.

Alcoholism is often associated with headache. In acute alcoholism, the headache probably results from cerebral hyperæmia; in chronic alcoholism it is often due to a low grade of meningitis.

Among other headaches of toxic origin may be mentioned those due to constipation, lead-poisoning, diabetes, infectious fevers, and absorption of foul gases.

Hysterical Headache.—In hysteria there is often a persistent headache, which grows worse at the menstrual periods, and which improves under pleasurable excitement. It may be diffuse, but frequently it is localized, and is described as resembling the effect which would be produced by a nail being driven into the head; hence it has been termed *clavus*.

Diagnosis.—Headache must be distinguished from *migraine*. In the latter there are usually prodromal symptoms, disturbances of vision, pupillary changes, and the pain is frequently confined to one side of the head.

Headache in the region of the orbit may be mistaken for

acute glaucoma, but in the latter condition the eye is inflamed; the cornea is hazy; the pupil is sluggish; vision is impaired; and on palpation the affected eyeball is found to be harder than its fellow.

TREATMENT.—In the interval between the attacks careful search should be made for the cause, which, if possible, must be removed. In the reflex headache of eye-strain the adjustment of proper glasses is often all that is required. In gastric headache, the associated catarrh of the stomach must be treated by a light diet and the use of such remedies as bismuth and nitrate of silver. In the headache of anæmia, a nutritious diet, with iron, arsenic, and other tonics will be required. In headaches of uræmic origin, a milk diet with measures calculated to increase the action of the skin, bowels, and kidneys, will often afford considerable relief. In malarial headache quinine in large doses with arsenic will effect a cure.

The Attack.—In headache dependent upon gastric acidity, after unloading the stomach with a non-irritating emetic, bromides with antacids will prove useful, thus :—

℞ Sodii bromid., ʒij;
Spt. ammon. aromat., fʒij;
Aquæ q. s. ad fʒiij.—M.
Sig.—A tablespoonful every hour or two.

In headache of acute cerebral congestion the feet should be soaked for ten or fifteen minutes in very hot water; an ice-bag placed on the head; and some sedative like the following administered :—

℞ Phenacetin, ʒj;
Sodii bromid., ℥ss.—M.
Ft. in chart No. xii.
Sig.—One powder every hour or two until relieved.

When the attack is very severe, aconite (gtt. j–ij) may be given every hour or two.

In cerebral anæmia good temporarily follows the use of antipyrin or phenacetin, especially in combination with caffeine, thus :—

℞ Phenacetin, ʒj;
Caffein. citrat., gr. xxiv.—M.
Ft. in chart No. xii.
Sig.—One as required.

In rheumatic headache salol is very useful; it may be combined with antipyrin:—

℞ Salol, ʒss;
Antipyrin, ʒj.—M.
Ft. in chart No. x.
Sig.—One every hour or two until relieved.

In uræmic headache the diet should be restricted to milk, action of the bowels secured by a saline draught, and diuresis encouraged by digitalis, caffeine, or the vegetable salts of potassium:—

℞ Potass. citrat., ʒij;
Spt. juniperi, fʒvj;
Æther. nitros., fʒij;
Infus. scoparii, fʒvj.—M. (Day.)
A wineglassful, thrice daily.

NEURITIS.

Definition.—Inflammation of nerves.

Etiology.—(1) It may result from traumatism—blows, wounds, or compression. (2) It may be due to exposure to cold and wet. (3) It may be secondary to inflammation of adjacent structures. (4) It may be secondary to rheumatism, gout, syphilis, or one of the infectious fevers.

Pathology.—The sheath, interstitial connective tissue, or fibres may be independently affected, but as a rule, all parts of the nerve are involved. When the process is acute the nerve is red and swollen, and microscopic examination reveals an infiltration of leucocytes, with more or less granular degeneration of the fibres.

In *chronic neuritis* the nerve-trunk is gray, shrivelled, and hard, and microscopic examination shows an overgrowth of connective tissue and granular degeneration of fibres.

Symptoms of Acute Neuritis.—There are three sets of phenomena—sensory, motor, and trophic.

Sensory Symptoms. — There is severe pain following the course of the affected nerve, which is tender to the touch. The pain is often associated with various manifestations of paræs-

thesia, such as burning, numbness, tingling, and the like. The part is at first hyperæsthetic, but later it is more or less anæsthetic.

Motor Symptoms.—Muscular power is impaired; there may be fibrillar tremors; and the reflexes are diminished or lost.

Trophic Symptoms.—An eruption of herpes sometimes follows the affected nerves. The skin may become glossy and the nails lustreless and brittle. In advanced cases there are wasting of muscles and impaired electro-contractility. Occasionally effusion into the joints is observed.

In some cases there may be febrile symptoms.

Chronic neuritis is characterized by pain, anæsthesia, paresis, atrophy and contracture of the muscles, reactions of degeneration, "glossy skin," and thickening and brittleness of the nails.

Diagnosis.—Neuritis may be mistaken for *neuralgia;* but in the latter the pain is paroxysmal and is unassociated with tenderness along the course of the nerve, paræsthesia, anæsthesia, paresis, and changes in the electro-contractility.

Prognosis.—In acute cases the prognosis is guardedly favorable; the duration is from a few days to several weeks. In chronic neuritis, after the development of marked trophic changes, the prognosis is grave.

Treatment.—The cause should be ascertained and, if possible, removed. In rheumatism, alkalies and salicylates are indicated. In syphilis, iodide of potassium should be administered in large doses. The part should be put at rest. For the pain, sedative lotions (lead-water and laudanum), warm fomentations, or small blisters may be applied to the affected parts, and morphine administered hypodermically. When morphine is contraindicated, salicylate of sodium or phenacetin may be employed in its stead. After the subsidence of acute symptoms, iodide of potassium may be given for its absorbent effect and small blisters applied locally. Restoration of power will be assisted by massage and electricity, and by the administration of strychnine, internally or hypodermically.

MULTIPLE NEURITIS.

Definition.—Inflammation of several nerve-trunks, resulting from a general cause, and characterized by pain, paræsthesia, anæsthesia, paresis, and muscular atrophy.

Etiology.—Alcoholism, syphilis, rheumatism, the infectious fevers, exposure to cold and wet, and mineral poisoning are common causes. In the Orient, multiple neuritis occurs as an endemic disease (Kakké or Beri-beri), which is probably microbic in origin.

Symptoms.—The acute form is characterized by a chill followed by moderate fever (102°–103°), headache, pain in the back, malaise, coated tongue, loss of appetite, constipation, febrile urine, and the following local phenomena: Pain, numbness, and tingling in the affected limbs; loss of power, especially in the legs and extensor muscles; abolition of the reflexes; atrophy of the muscles; more or less anæsthesia; and tenderness over the nerve-trunks.

Chronic Form.—Febrile symptoms are absent and the disease is manifested by pains in the limbs, hyperæsthesia, paræsthesia, irregular areas of anæsthesia, loss of power, abolition of the deep reflexes, tenderness over the nerve-trunks, wasting of the muscles, impaired electrical contractility, and œdema of the hands and feet.

Complications.—Delirium, delusions, and hallucinations are not uncommon, especially in the alcoholic variety. The disease is sometimes associated with locomotor ataxia.

Diagnosis. *Locomotor Ataxia.*—The absence of the lightning-pains, girdle sensation, Argyll-Robertson pupil, and the presence of paralysis, wasting, and neural tenderness will serve to distinguish multiple neuritis from locomotor ataxia.

Prognosis.—Guardedly favorable. Acute neuritis sometimes proves fatal from involvement of the respiratory muscles. In chronic cases of long duration the outlook is not hopeful.

Treatment.—Acute cases should be kept at absolute rest. For the relief of pain hot fomentations, lead-water and laudanum, and rubefacient liniments may be applied to the affected limbs; and morphine, antipyrin, phenacetin, or salicylic acid

administered internally. After acute symptoms have subsided, massage, electricity, and Swedish movements should be employed to secure a return of power. An ointment of mercury and belladonna may be used for its absorbent and anodyne effect. Strychnine hypodermically is an invaluable muscular tonic. Rigidity is best relieved by manipulation and the frequent use of warm baths. In syphilitic cases employ mercurial inunctions and iodide of potassium.

SCIATICA.

Definition.—Pain along the sciatic nerve, usually resulting from neuritis.

Etiology.—Male sex, middle life, gout, rheumatism, and syphilis are predisposing causes. Exposure to cold and wet is the common exciting cause. Very rarely sciatica is a secondary condition resulting from the presence of an intra-pelvic growth or from caries of the bone in joint disease.

Symptoms.—The disease may begin abruptly or gradually, and is characterized by a sharp shooting pain running down the back of the thigh. Movement of the limb intensifies the suffering. The pain may be uniformly distributed along the course of the nerve, but not infrequently there are certain spots where it is more intense. Subjective sensations, such as tingling and numbness, are often noted. The nerve may be extremely sensitive to touch. The symptoms grow worse at night and on the approach of stormy weather. The duration of the attack varies from a few days to several months. In long-standing cases the muscles become atrophied and rigid.

Diagnosis. *Coxalgia.*—In this affection the pain is most marked in the hip- and knee-joints; pressure over the trochanter elicits pain; and the nerve is not tender to the touch.

Prognosis.—Recovery follows in the majority of cases when treatment is instituted early and is persistently carried out. In some cases relapses occur frequently, and finally the pain becomes more or less continuous.

Treatment.—In the acute stage rest is essential. Hot fomentations or linear blisters may be applied along the course of the nerve. Deep injections of morphine, antipyrin,

or cocaine may be required to relieve the pain. In rheumatic cases full doses of the salicylate of sodium are very useful. In chronic cases prolonged rest is desirable. Counter-irritation should be made by frequent small blisters, by the actual cautery, or by acupuncture. Deep injections along the course of the nerve give much relief, and one of the following remedies may be so employed: morphine and atropine, cocaine, antipyrin, or plain water. Electricity sometimes does good. Internally iodide of potassium in small doses is useful; in syphilitic cases it should be given in large doses. The following combination is also efficient:—

℞ Tinct. aconiti rad.,
Tinct. colchici sem.,
Tinct. belladonnæ,
Tinct. cimicifugæ, āā fʒij.—M. (METCALF.)
Sig.—Twelve drops every four to eight hours.

FACIAL PARALYSIS.

(Bell's Palsy.)

ETIOLOGY.—Paralysis of one side of the face may result: (1) From a tumor, clot or abscess involving the facial centre on the cortex of the brain or the nucleus of the facial nerve; (2) from the pressure of inflammatory exudate on the nerve-trunk between the brain and the skull; (3) from paralysis of the nerve within the petrous portion of the temporal bone, excited by a fracture, or by an extension of inflammation of the middle ear; (4) from inflammation of the peripheral filaments, excited by exposure, injury, rheumatism, or one of the infectious fevers.

SYMPTOMS.—The side affected is expressionless; the natural lines are obliterated; the angle of the mouth droops; the eye cannot be closed; tears flow over the cheek; and speech is affected from an inability to pronounce the labials. When the patient attempts to laugh or whistle, the absence of movement on the affected side becomes still more conspicuous. In peripheral neuritis the reflexes are abolished; and when the nerve is involved in the temporal bone there may be a loss of taste in the anterior part of the tongue.

Diagnosis.—When the lesion is in the brain the paralysis is rarely complete, the upper part of the face usually escaping; neighboring cranial nerves are frequently affected; and other evidences of organic brain disease are generally present.

When the nerve is involved within the Fallopian canal there is often a loss of taste in the anterior part of the tongue, and some disturbance of hearing—deafness or perhaps hypersensitiveness to sound.

In peripheral neuritis the history, the completeness of the paralysis, the absence of reflexes, and the presence of the reactions of degeneration will assist in the recognition of the lesion.

Prognosis.—The prognosis will vary with the cause. It should be guardedly favorable when the paralysis is due to peripheral neuritis.

Treatment.—The cause should be ascertained, and if possible, removed. In paralysis of centric origin little can be done, except in syphilitic cases, In middle-ear disease remedies should be directed to that organ. When paralysis results from inflammation of the peripheral filaments of the facial nerve, blisters should be applied near the stylo-mastoid foramen, and as it often appears to be an expression of rheumatism, salicylates may be given internally. Later, a course of iodide of potassium will be useful, and restoration of power may be materially assisted by massage, electricity, and local injections of strychnine.

EPILEPSY.

(Idiopathic Epilepsy, Falling Sickness.)

Definition.—A chronic disease of the nervous system, characterized by paroxysms of unconsciousness which are usually associated with general convulsions.

Etiology.—Heredity predisposes, and the ancestral disease may not have been epilepsy but insanity, hysteria, or another neurosis. It generally begins before puberty, and very rarely after the twenty-fifth year. All causes which impair the health and exhaust the nervous system exert a predisposing influence. The reflex convulsions of children resulting from gastric irritation, worms, etc., if long continued may induce

chronic epilepsy. In these cases, although the exciting cause has been removed, the habit of spontaneous motor discharge, through constant repetition, is established, and may continue through life. In those subject to convulsions, overwork, gastric irritation, or excitement may precipitate an attack.

Pathology.—No demonstrable causal lesions are detected. The disease apparently depends upon an instability of the motor centres, so that from trivial exciting causes violent discharges occur from time to time.

Symptoms. *Grand Mal.*—The seizure is often preceded by a peculiar sensation termed an *aura*, beginning in a finger or toe and rising until it involves the head, when the patient gives a shrill scream and falls to the floor unconscious. At first the face is pale, the pupils contracted, and the body thrown into a tonic spasm in which the head is retracted and rotated, the limbs forcibly extended, and the thumbs turned into the palms and firmly clenched by the flexed fingers. In a few seconds the tonic spasm relaxes, the movements become clonic or intermittent, the pupils dilated, the face cyanosed, and from the violent contraction of the masseters frothy saliva, often blood-streaked, pours from the mouth. The clonic spasms continue for a minute or two, and are generally followed by a period of coma lasting from a few minutes to several hours. Sometimes the patient returns at once to consciousness, and complains simply of weakness, muscular soreness, and mental confusion. More rarely the convulsion is followed by an outbreak of mania, or of *epileptic automatism*, a condition in which the patient unconsciously performs simple or complicated acts.

Petit Mal.—In this type the seizure consists of momentary unconsciousness, with pallor, and rarely twitching of the muscles. The patient suddenly stops in the midst of his work or conversation, remains quiet for a few seconds, and then continues where he left off, perhaps unconscious of the interruption. *Petit mal* may be a forerunner of *grand mal* or may alternate with it.

Between these two extremes, the seizures manifest all grades of severity. The frequency of the paroxysms varies considerably; they may occur as seldom as once a year, or as often as

ten or twelve times a day. A marked periodicity in their recurrence is often observed.

The term "status epilepticus" is applied to a series of convulsions which follow each other in rapid succession, and which are associated with high fever.

The epileptic may manifest no other symptoms beyond the convulsions, but when the latter are very frequent the health fails and the mental power deteriorates.

Diagnosis.—The convulsions of idiopathic epilepsy must be distinguished from those due to *organic brain disease* (organic epilepsy). The latter affection rarely develops before twenty-five; the aura may be connected with the special senses, which is uncommon in idiopathic epilepsy; the convulsion is often confined to one member or to one side of the body, and may not be associated with unconsciousness (Jacksonian epilepsy); the convulsion may begin in one member and then become generalized; and finally, in a large proportion of the cases of organic epilepsy, there will be a history or concomitant symptoms of syphilis, or the evidence of cerebral injury.

Uræmia.—Uræmic convulsions may be recognized by the history and the results of the urinary analysis.

Prognosis.—Generally unfavorable. Arrest of the disease is rare, but amelioration is often secured by treatment.

Treatment. *Preventive.*—Careful search should be made for the cause which excites the paroxysms; this will often be found in some disturbance of the gastro-intestinal tract. The diet should be light, and as a rule, largely vegetable. Constipation must be relieved by diet, exercise, or the use of mild laxatives. Undue mental and physical excitement should be avoided. Systematic exercise and frequent bathing followed by friction of the skin lessen the sensitiveness of the nervous system. The most reliable drugs are the bromides; one or two drachms of a combination of the bromides of sodium, potassium, and ammonium may be given daily. Strontium bromide is often efficacious, and it is less depressing than the other bromides. The tendency to acne may be considerably lessened by the addition of a drop or two of Fowler's solution with each dose. A small amount of antipyrin often lessens the amount of the bromide required to check the convulsions.

℞ Ammon. bromid., ʒvj ;
Antipyrin, ʒj ;
Liq. potass. arsenitis, fʒj ;
Aq. menthæ pip., q. s. ad fʒvj.—M. (Wood.)
Sig.—Tablespoonful in water night and morning.

When the bromides fail, one of the following remedies may be employed: oxide of zinc (gr. vj–xv a day), picrotoxin (gr. $\frac{1}{100}$ thrice daily), sulphonal, borax, or belladonna.

When an *aura* gives warning of a seizure, the inhalation of nitrite of amyl may abort it.

Surgical interference is indicated in Jacksonian epilepsy, and in those cases in which the convulsion begins in one member and subsequently becomes generalized.

The Attack.—As the seizure is short, special treatment is rarely required. Injury of the tongue may be prevented by placing a piece of cork between the teeth. In the *status epilepticus* chloroform or nitrite of amyl may be administered by inhalation, and hyoscine (gr. $\frac{1}{100}$) or morphine given hypodermically.

APHASIA.

Definition.—A failure of word-memory ; an inability to utter words, to comprehend them, or to write them.

Varieties.—Sensory and motor.

Sensory Aphasia. *Word-blindness.*—Inability to recognize written or printed words. It results from a lesion of the angular and inferior parietal gyri of the left side.

Word-deafness.—Inability to interpret spoken words. The lesion is in the posterior half of the first and second temporal convolutions of the left side.

Amnesic Aphasia (*Concept Aphasia*).—Inability to recall words. The lesion is in the conducting paths between the receptive and emissive centres of the brain, probably in the third left temporal convolution.

Apraxia (*Psychic Blindness*).—Inability to interpret perceptions of sight (mind-blindness) ; of smell (mind-anosmia) ; of taste (mind-ageustia) ; of hearing (mind-deafness) ; or of touch (mind-ataetilia). In mind-blindness the lesion is in the supra-marginal and angular gyri.

Motor Aphasia. *Aphemia, or Ataxic Aphasia.*—Inability

to utter words, though knowing their meaning. The lesion is in the posterior part of the third left frontal convolution (Broca's region).

Paraphasia (*Conduction Aphasia*).—The misuse of words or syllables. It is due to defect in the tracts associating the cortical speech centres.

Motor Agraphia.—Inability to write words from a lack of muscular coördination, rather than a loss of power. It is frequently associated with hemiplegia of the right side. The lesion is in the posterior part of the mid-frontal convolution (?).

Amimia.—Inability to express thoughts by signs. This condition, which may be regarded as a form of aphasia, may be sensory or motor. It is frequently dependent upon a lesion of the left third frontal convolution.

Paramimia.—A misuse of the signs intended to convey thought. It should be regarded as a form of conduction aphasia.

Pathology.—The lesions which produce aphasia are manifold; the most important are: Tumor, gumma, abscess, depressed fracture, embolism, thrombus, or softening in the localities which correspond to the various forms of aphasia. In right-handed subjects the lesion is on the left side of the brain; in the left-handed it may, however, be on the right side. Aphasia is not always due to organic disease; it may be noted in congestion of the brain, in sudden fright, in the convalescence of fevers, in migraine, after epileptic seizures, and in hysteria.

Diagnosis.—Aphasia must be distinguished from *aphonia.* The latter condition is an inability to utter sounds, a power not lost in aphasia; moreover, aphonia is generally dependent upon some abnormality of the larynx or of the nerves leading thereto.

Prognosis.—This depends entirely on the cause. After apoplexy the prognosis should be guarded. In cerebral softening it is absolutely unfavorable. When aphasia develops in the young the outlook is much more hopeful.

Treatment.—The causal condition will require attention. The patient may be instructed to speak and to interpret after the manner employed in teaching the young.

VERTIGO.

(Dizziness, Giddiness, Swimming in the Head.)

DEFINITION.—A sense of unstable equilibrium in which the patient himself or surrounding objects appear to be in a state of rapid oscillation or rotation. It is a symptom of many conditions.

ETIOLOGY.—Vertigo may result from :—

1. Cerebral anæmia or congestion. The dizziness preceding a fainting fit is an illustration of the former, and that following exposure to the rays of the sun is an illustration of the latter. Vertigo is often a pronounced symptom of chronic cerebral congestion and anæmia. The vertigo of chronic heart disease and of neurasthenia is included under this head.

2. Reflex irritation. The most common example of this form is the vertigo dependent upon gastric disturbances. It is also noted in eye-strain, uterine disease, constipation, and disease of the internal ear. The last is termed *labyrinthine vertigo*, or *Ménière's disease*, and has been described elsewhere.

3. Organic disease of the brain and cord. Cerebral tumor, meningitis, and softening are frequently associated with vertigo. It is often quite marked in cerebellar disease. It may be a pronounced symptom in disseminated sclerosis and locomotor ataxia.

4. Toxic substances in the blood. The vertigo observed in lithæmia, uræmia, and diabetes is included under this head. When taken in large doses, certain drugs, as alcohol, belladonna, cannabis indica, lobelia, and conium, may produce the symptoms. It is often a marked symptom of chronic lead-poisoning.

5. Epilepsy. Vertigo may precede, follow, or take the place of an epileptic seizure.

6. Hysteria. Occasionally marked vertiginous attacks are connected with hysteria.

7. Unknown causes. The term *essential vertigo* has been applied to those cases in which, after the most exhaustive study, no adequate cause can be ascertained. There is sometimes an hereditary tendency to this form of vertigo.

DIAGNOSIS.—Vertigo must be distinguished from *pepit mal*,

or *minor epilepsy*. The history, the presence of a definite cause, and the absence of unconsciousness and of convulsive movements will serve to separate vertigo from epilepsy.

The determination of the cause of the vertigo must be based upon the history, the age at which it develops, and a critical examination of the various organs.

Prognosis.—This will depend entirely on the cause; when the latter can be removed, the prognosis is favorable.

Treatment.—This must be directed to the causal condition.

MÉNIÈRE'S DISEASE.

(Labyrinthine Vertigo, Aural Vertigo.)

Definition. — Paroxysmal vertigo, probably depending upon disease of the internal ear.

Etiology and Pathology. — The exact cause of Ménière's disease is still undetermined. In some cases, however, inflammatory changes have been observed in the semicircular canals. Very severe acute attacks are sometimes observed in patients previously healthy. In these the lesion is probably an active hyperæmia of, or a hemorrhage into, the labyrinth. It is probable that mild forms of the disease can be indirectly induced by lesions of the middle ear.

Symptoms. — Frequently prodromes precede the attack, such as deafness or earache. These, however, may be absent, and the attacks ushered in with extreme vertigo and *tinnitus aurium*. The latter is often compared to the escape of steam, the buzz of an insect, or the discharge of a cannon. The patient feels as if he or surrounding objects were being whirled violently around, and in severe cases the face is pale and anxious; the surface is clammy; there are nausea and vomiting; and the patient falls unconscious.

As a rule, there is deafness in one ear at least, but exceptionally, hearing may be quite normal. At first the paroxysms may occur at long intervals, but as the disease advances they become more frequent and the tinnitus and deafness become more marked.

Diagnosis.—The paroxysmal vertigo, deafness, and *tinnitus aurium* are the diagnostic features.

PROGNOSIS.—The prognosis should always be guarded. Some cases recover entirely, but in the majority the vertiginous attacks continue until the deafness in the affected ear becomes complete.

TREATMENT.—The middle ear should be carefully examined, and any existing disease treated. Severe counter-irritation by blisters, or the actual cautery applied behind the ear, may be of some service. Bromide of potassium or large doses of hydrobromic acid may give temporary relief. Charcot recommends quinine in sufficient doses to cause cinchonism.

HYSTERIA.

DEFINITION. — Hysteria is a functional disease of the nervous system, manifested by symptoms of the most varied character, and associated with impaired will-power and increased impressionability.

ETIOLOGY.—Females are especially predisposed, although it occasionally develops in males. It is most common in early adult life. The chief causal factor is heredity, the disease frequently being transmitted through hysterical, epileptic, or insane parentage.

Traumatism, defective education, prolonged emotional excitement, such as worriment, anxiety, disappointment and grief, and all causes which lower the vitality serve to excite it in susceptible individuals.

PATHOLOGY.—No causal lesions can be detected after death.

SYMPTOMS.—The various manifestations may be described under three heads: (1) Motor, (2) sensory, and (3) psychical.

Motor Phenomena.—Paralysis not infrequently results from hysteria; it may take the form of a hemiplegia, paraplegia, or monoplegia, although the first is by far the most common. The paralysis is generally paroxysmal, and is frequently associated with contractures and anæsthesia. The affected muscles do not waste.

Local paralysis is also common; thus there may be aphonia from paralysis of the vocal cords; dysphagia, from paralysis of the œsophagus; and incontinence of urine, from paralysis of the bladder.

Convulsive seizures are common manifestations of hysteria, and may closely simulate the paroxysms of true epilepsy; but there is no aura; the patient usually falls in a comfortable place; consciousness is only apparently lost, for after the seizure she remembers all that has transpired; the tongue is rarely bitten; the eyes are partially closed; the face is expressive of some emotion; screaming or sobbing is of frequent occurrence; the movements are apt to be tonic, so that the patient assumes the position of opisthotonos, or if clonic, they are apt to be violent and purposive; the seizures are of long duration, and may be continued for several hours or days, and firm pressure over the ovaries may exaggerate or re-excite them.

The spasms may be local; thus there may be retention of urine, from spasm of the bladder; asthma, from spasm of the bronchi; hiccough, from spasm of the diaphragm; persistent vomiting, from spasm of the stomach; dysphagia, from spasm of the œsophagus; and a "phantom tumor," from spasm of abdominal muscles associated with flatulent distention of the intestines.

Among other motor phenomena may be mentioned obstinate tremors, choreiform movements, and contractures of certain groups of muscles.

Sensory Phenomena.—There may be a complete loss of sensation in certain parts, as one side of the body. Anæsthesia without other nervous phenomena is usually hysterical. In some cases tactile sensation is preserved and there is a loss only of thermic or painful sensations. The anæsthetic part is often unusually pale, and when pricked with a needle fails to bleed (ischæmia).

The special senses may be involved; thus there may be contraction of the field of vision, complete blindness, loss of smell, loss of taste, or loss of hearing. These special-sense palsies are usually transient, and often alternate with one another.

Instead of anæsthesia, there may be hyperæsthesia or pain. Severe pain in the stomach may simulate gastralgia. An exquisitely painful and tender condition of the abdomen may be mistaken for peritonitis. A localized pain in the head, described as resembling the effect of a nail being driven into it, is termed hysterical *clavus*. The joints sometimes become swollen and very tender, resembling arthritis (neuromimesis).

Intense pain over the heart may simulate angina pectoris. The spine is often the seat of hyperæsthesia, especially in spots, and this *spinal irritation* is often associated with pain in parts corresponding to the distribution of nerves which have their origin in the hyperæsthetic area.

A very common abnormal sensation is the globus hystericus, *i. e.*, a feeling as of a ball rising in the throat and impeding respiration.

Psychical Phenomena.—Frequently the only conspicuous mental phenomenon is the great lack of will-power; but generally the patients are more or less excitable, highly mercurial, and easily moved to laughter or tears. They frequently manifest a great fondness for sympathy, and this, in connection with their weak will-power and lowered moral tone, often leads them to feign symptoms which they really do not have. Among the more serious mental manifestations may be mentioned delirium, ecstasy, catalepsy, and trance.

Diagnosis.—The recognition of hysteria is often attended with great difficulty, especially as it is frequently associated with symptoms which really have an organic basis. In making a diagnosis, the history, sex, and temperament must be carefully considered. The manifestations usually develop abruptly; are generally paroxysmal; appear without obvious cause; often subside spontaneously under some emotional excitement; rarely lead to any impairment of the health; and are usually associated with a history of other hysterical phenomena.

Prognosis.—As regards life the prognosis is good. In rare instances death has followed exhaustion induced by repeated convulsions or prolonged fasting. While hysteria usually ends in recovery, the duration of the illness is a matter of great uncertainty.

A speedy recovery is to be expected in those cases where the hysterical phenomena are connected with some obvious cause which can be removed.

Treatment.—Careful search should be made for some exciting cause, which, if found, should be removed when possible. The physical condition is generally reduced, and careful study must be given to the diet, exercise, amusement, clothing, etc., with the view of improving it. Tonics like

iron, arsenic, strychnine, hypophosphites, cod-liver oil, and malt are often indicated, and they may be advantageously combined with such nerve sedatives as valerian, asafœtida, sumbul, and the like; in the milder manifestations, the following pill may prove useful:—

℞ Acid. arsenosi, gr. ½;
Ferri sulph. ex.,
Ext. sumbul, āā gr. xx;
Asafœtidæ, gr. xl.—M. (Goodell.)

Ft. in pil. No. xx.
Sig.—One after each meal.

Or—

℞ Quinin. valerianat.,
Zinci valerianat.,
Ferri valerianat., āā gr. xxiv.—M.

Ft. in pil. No. xxiv.
Sig.—One, thrice daily.

℞ Auri et sodii chloridi, gr. v;
Tragacanth., ʒj;
Sacchari, q. s.—M. (Mills.)

Ft. in pil. No. xl.
Sig.—One thrice daily, increased to three thrice daily.

The more thoroughly the physician is able to inspire confidence and to control his patient, the more likely is he to effect a cure. Firmness tempered with kindliness and encouragement is essential to success.

While hypnotism appears to have been somewhat useful in France, in this country, although employed but to a limited extent, it has not given encouraging results, and moreover, in the event of failure, seems capable of aggravating the hysterical condition.

In long-continued convulsive seizures, cold water may be dashed on the face and chest, or hyoscine administered hypodermically. In obstinate cases an anæsthetic should be employed. In the various form of paralysis electricity is often useful. In some cases static electricity, no doubt from the profound mental effect which it has induced, has given excellent results.

In aggravated cases the "rest-cure" introduced by S. Weir Mitchell is often applicable. It consists in isolation from

sympathizing friends and relatives; abundant feeding, especially with milk; and complete rest of body and mind with passive exercise obtained by massage and electricity.

NEURASTHENIA.

(Nervous Prostration.)

DEFINITION.—A term applied to a group of symptoms apparently resulting from exhaustion of the nerve-centres.

ETIOLOGY.—An hereditary tendency, prolonged mental work, or emotional excitement, excesses, defective education, traumatism, and irregular living are general predisposing factors.

SYMPTOMS. *Cerebral Symptoms.*—Depression of spirits, indisposition, inability to concentrate the mind on one subject for any length of time, insomnia, vertigo, headache, irritability of temper, introspection, and morbid fears.

Spinal Symptoms.—Sometimes these predominate, when the condition is termed *spinal irritation*, and its chief manifestations are: Pain in the back, spots of tenderness along the spine, weakness of the extremities, great prostration after moderate exertion, and various subjective phenomena, such as numbness, tingling, formication, and neuralgic pains.

Gastro-intestinal Symptoms.—Anorexia, coated tongue, and constipation.

Circulatory Symptoms.—Palpitation, tachycardia, pseudo-angina, cold extremities, and sometimes violent pulsation of the aorta.

Sexual Symptoms.—In females, amenorrhœa or dysmenorrhœa; in males, impotence or spermatorrhœa.

DIAGNOSIS.—The diagnosis is rarely difficult. Before relegating a case to this class, care must be taken to exclude *organic* disease, and *such general disorders as lithæmia* and *anæmia*.

Hysteria.—This affection may be distinguished by the abrupt onset, the intermittent character of the symptoms, and the presence of paralysis, anæsthesia, convulsions, or the globus hystericus.

Prognosis.—When the cause can be removed and the patient controlled, the prognosis is favorable.

Treatment.—The treatment is largely hygienic and dietetic, and will vary considerably in different cases. Where there has been inactivity, regulated physical exercise will be of great value; on the other hand, the weak and anæmic will require rest. In the latter case, the plan of treatment introduced by S. Weir Mitchell, and known as the "rest-cure," often gives brilliant results. In all cases careful attention must be given to the diet, bathing, and clothing, and the patient assured that he is suffering from no incurable disease. Frequent bathing with salt water, followed by friction of the skin, will often add to the general vigor. Tobacco and alcohol must be interdicted, and tea and coffee used very sparingly. Tonics like iron, arsenic, quinine, strychnine, and phosphorus are often indicated.

CHOREA.

(Chorea Minor, St. Vitus's Dance.)

Definition.—A nervous affection occurring especially in children, and characterized by irregular movements which increase under excitement and cease during sleep.

Etiology.—Childhood (between five and fifteen), female sex, season (spring), nervous temperament, and the rheumatic diathesis are general predisposing factors. It sometimes develops suddenly after mental or emotional excitement, such as anxiety, fear, or grief. It may be excited by reflex irritation, as an adherent prepuce, intestinal parasites, etc. It not infrequently develops in the course of pregnancy.

Pathology.—It is customary to look upon chorea as a neurosis, since no constant lesions have been discovered to account for its clinical manifestations. In some cases endocarditis, and emboli in the minute cerebral vessels have been discovered, but their relation to chorea has not yet been determined. A microbic origin has been suggested.

Symptoms.—The first manifestations are usually restlessness and awkwardness in movement. The child cannot remain still, but is constantly raising its shoulders, jerking its head,

twisting its fingers, or shuffling its feet. Frequently these symptoms develop so insidiously that the disease is not recognized, and the child is punished for being fidgety.

When the disease is fully established the disorderly movements become more marked, and may be confined to one member or may involve the entire body. When the facial muscles are affected, the most grotesque expressions are produced; involvement of the arms may interfere with eating and dressing; when the legs suffer the gait becomes jerking and stumbling; involvement of the larynx causes stammering; and spasm of the muscles of deglutition induces difficult swallowing and choking-spells. When the attention is directed to the movements they invariably grow worse, but they diminish during repose and cease entirely during sleep. Sometimes, in addition to the involuntary movements, there is a distinct loss of power in the affected members. The general health is usually more or less impaired. The child is anæmic; the temper is irritable; and the mental power deficient. Auscultation of the heart often detects a murmur which may be either an expression of anæmia or of complicating endocarditis.

In some cases (*chorea insaniens*) the movements are so violent that the patient is unable to walk, eat, or even to lie down. Fever develops, and ultimately the mind becomes delirious. Death frequently results from exhaustion. This form is usually observed in adults, and especially in primiparæ.

Diagnosis.—The recognition of chorea is rarely attended with difficulty. *Disseminated spinal sclerosis* may be distinguished by the presence of nystagmus, a scanning speech, increased reflexes, and a rhythmical tremor which is only excited by movement.

Prognosis.—In simple chorea recovery usually follows in the course of two or three months. Death from heart complications is a rare termination. Relapses are not infrequent. Among the possible sequelæ may be mentioned imbecility and chronic chorea.

Chorea insaniens frequently terminates fatally through exhaustion.

Treatment.—Rest of body and mind is an essential element of the treatment. The child should be taken from

school and placed under the most favorable hygienic conditions. Careful search should be made for reflex irritation, such as adherent prepuce, intestinal parasites, eye-strain, etc. All excitement must be avoided. Amusement in the open air when the weather is fine is to be recommended. As the child is generally anæmic, iron is indicated in the majority of cases. Among the special remedies arsenic holds the first place. Fowler's solution may be given in doses of two drops thrice daily, gradually increased to eight or ten drops thrice daily. Among other remedies may be mentioned the fluid ext. of cimicifuga (♏x increased to ʒj thrice daily), hyoscyamine (gr. $\frac{1}{150}$–$\frac{1}{100}$), and quinine (gr. iij–v every two or three hours).

In *Chorea insaniens* forced feeding should be resorted to. Morphine and other sedatives may be employed hypodermically. Chloroform may be required to control temporarily the movements. Severe cases of chorea complicating pregnancy will call for the induction of premature labor.

PARALYSIS AGITANS.

(Parkinson's Disease, Shaking Palsy.)

Definition.—A chronic nervous disease, characterized by a fine, slowly-spreading tremor, muscular weakness and rigidity, and a peculiar gait, termed *festination*.

Etiology.—Advanced life, a neuropathic tendency, mental strain, heredity, and exposure to cold and wet are predisposing factors. It sometimes develops suddenly after intense mental or emotional excitement.

Pathology.—The pathology is unknown. The lesions found—degeneration of arterioles, perivascular sclerosis, pigmentation of ganglionic cells—are similar to those induced by senility.

Symptoms.—In some cases the onset is abrupt, but more commonly the disease develops insidiously. The first symptom is usually a fine tremor beginning in the hand or foot, which may slowly spread until it involves all the members; the head is rarely affected. At first the tremor may be paroxysmal, but as the disease advances it becomes almost continuous. Excitement increases it, but it is noteworthy that physical effort temporarily diminishes or checks it The face becomes

expressionless, and the speech slow and measured. Later, muscular rigidity develops; the head is bowed, the body bent forward, the arms flexed, the thumbs turned into the palms and grasped by the fingers, and the knees slightly bent. At this time the gait is characteristic: the steps grow faster and faster, the body inclines more and more forward until the patient falls, seeks support in some neighboring object, or straightens himself by a supreme effort of the will. The term *festination* has been applied to this peculiar gait. Occasionally a tendency to fall backwards—*retropulsion*—replaces festination. The rigidity and muscular weakness render all movements slow and labored.

Intelligence is usually good. There is no anæsthesia, but there are various manifestations of paræsthesia, such as numbness and tingling; a sensation of heat is especially noted. In some cases free perspiration has been observed.

Diagnosis.—The tremor, rigidity, weakness, flexion of the body and members, lack of facial expression, and *festination* are the diagnostic features. In some cases the tremor is absent. Paralysis agitans must be distinguished from *disseminated sclerosis*. In the latter the tremor is coarse, is frequently absent when the patient is quiet, and is made worse by efforts to control it; cerebral symptoms are generally present; nystagmus is often noted; and the attitude and gait are entirely different from those of paralysis agitans.

Prognosis.—Recovery rarely, if ever, occurs. In some cases, after reaching a certain point, the disease remains stationary. The progress is slow and the duration indefinite.

Treatment.—Measures intended to improve the tone of the system are indicated; these are: A regulated diet, rest of body and mind, frequent bathing followed by friction of the skin, and the use of such tonics as iron, arsenic, and phosphorus. The rigidity and tremors are sometimes improved by massage and electricity. Among the remedies recommended for the tremors are bromide of potassium, hyoscyamine (gr. $\frac{1}{100}$), and hyoscine (gr. $\frac{1}{125}$), but the improvement following their use is only slight and temporary.

ARTISANS' CRAMP.

DEFINITION.—A spasmodic affection of the muscles induced by prolonged work requiring delicate coördination, and occurring only in the performance of that particular work.

ETIOLOGY.—It is more common in men than in women, and the nervous temperament predisposes to its development. The occupations in which it is most apt to occur are writing, piano-playing, sewing, and telegraphing.

PATHOLOGY.—The disease is evidently not peripheral, for when the other hand is substituted the condition soon develops in that member. It is probably dependent upon unnatural irritability of the nerve-centres.

Writers' Cramp.

(Graphospasm, Scriveners' Palsy.)

SYMPTOMS.—The condition usually begins with a sense of fatigue, weight, or actual pain in the affected muscles. Soon the fingers are seized with a tonic or clonic spasm whenever the pen is grasped (spastic form). In some cases the hand when put into use becomes the seat of a decided tremor (tremulous form); in a third group of cases the chief phenomena are excessive weakness and fatigue, which disappear as soon as the pen is laid aside (paralytic form).

PROGNOSIS.—Guardedly favorable. The disease is obstinate, but cure generally follows protracted rest.

TREATMENT.—Absolute rest is the essential element of treatment. The general condition should be improved by iron, arsenic, strychnine, and cod-liver oil. Massage, electricity, and passive movements give good results.

TETANY.

(Tetanilla, Intermittent Tetanus.)

DEFINITION.—A nervous affection, characterized by tonic spasms which are usually paroxysmal and involve the extremities.

ETIOLOGY.—It is most frequently observed in the young. In women it is frequently associated with pregnancy or lacta-

tion. It is sometimes excited by exposure, emotional excitement, or one of the infectious fevers. An epidemic form has been described, but some of the outbreaks seem to have been hysterical. A very grave form has been induced by thyroidectomy and by lavage in gastric dilatation.

SYMPTOMS.—The patient is seized with bilateral tonic spasms in the arms and legs. The jaws are rarely involved. The contractions are usually paroxysmal and are attended with pain. As was pointed out by Trousseau, they can be induced by pressure over the arteries and nerves of the affected limb. The electro-contractility of the muscles is greatly exaggerated. There may be slight œdema. Sensation is not disturbed; the mind is clear; and fever is slight or entirely absent.

DIAGNOSIS.—*Hysteria* may be distinguished from tetany by the history, the unilateral character of the contractions, the absence of muscular excitability and of Trousseau's sign.

Tetanus.—In this disease the spasms are continuous and early involve the jaws and trunk.

PROGNOSIS.—Usually favorable. Attacks following thyroidectomy and lavage sometimes prove fatal.

TREATMENT.—Good hygiene; tonics; electricity; sedatives like bromide of potassium, belladonna, and chloral. Warm or cold baths, followed by friction.

THOMSEN'S DISEASE.

(Congenital Myotonia.)

DEFINITION.—A disease confined to certain families, and characterized by tonic spasms of the muscles, induced by voluntary movements.

ETIOLOGY.—The disease is usually congenital, and transmitted from one generation to another. Several members of the same family are commonly affected.

PATHOLOGY.—Unknown.

SYMPTOMS.—The disease appears in early childhood, and is manifested by a tonic spasm of the muscles every time they are put in use; this is especially marked after periods of inactivity. In a few moments the rigidity wears away and the movements become free. From repeated contractions the

muscles become firm and extremely well developed. Under electrical stimulation the muscles contract and relax slowly.

Prognosis.—Incurable.

Treatment.—The condition improves under physical exercise.

RAYNAUD'S DISEASE.

(Symmetrical Gangrene.)

Definition.—A vaso-motor neurosis, characterized by local anæmia, congestion, or gangrene.

Etiology.—The cause is unknown. The disease probably consists in a local spasm or paresis of the vessels.

Symptoms.—In one form the part, usually the finger, becomes extremely pale, cold, and anæsthetic (local syncope). After a variable time these phenomena disappear and are followed by redness, heat, and tingling; such attacks may be excited by cold, and come and go without damaging the part.

In another form the affected part becomes swollen, dark red, and painful (*local asphyxia*), and if the attack persists bullæ may appear and gangrene develop. The gangrenous areas are often symmetrical, involving a finger on each hand, a toe on each foot, or both ears. Hæmoglobinuria may occur in, or replace, an attack.

Prognosis.—The attacks persist, but life is not endangered. In rare instances extensive gangrene develops and is followed by death.

Treatment.—Patients liable to attacks should be well protected against cold. Tonics are often indicated. Frequent bathing followed by friction is useful. Raynaud advises the use of a continuous current, one pole over the spine and the other over the affected area. Nitro-glycerine may prove useful.

ACUTE ANGIO-NEUROTIC ŒDEMA.

Definition.—A neurosis characterized by transient circumscribed œdema developing without obvious cause.

Etiology.—Beyond a distinct hereditary tendency nothing is known of its cause. According to Quincke, there is a tem-

porary vaso-motor dilatation of the vessels followed by the transudation of serum.

Symptoms.—Œdematous swelling suddenly appears in some part of the body, particularly in the face and hands. Coincident with the œdema there may be marked gastro-intestinal symptoms such as vomiting, gastralgia, and colic. The disease is allied to urticaria and the latter may precede the outbreak.

The attacks may occur at intervals of a few weeks.

Prognosis.—The peculiar tendency persists; unless the larynx is involved, it is unattended with danger.

Treatment.—General tonics, like iron, quinine, and strychnine are sometimes useful.

FACIAL HEMI-ATROPHY.

(Unilateral Progressive Atrophy of the Face.)

Definition.—A rare affection, characterized by progressive wasting of tissues—bones and soft parts—on one side of the face.

Etiology.—The disease usually develops in childhood. It has been excited by injury of the face.

Pathology.—In the few cases examined chronic trigeminal neuritis or lesions of the Gasserian ganglion have been discovered.

Symptoms.—The first phenomenon is often discoloration of the skin; this is soon followed by a slow wasting of all the tissues on the affected side of the face. The hair falls, the eye recedes, and the teeth drop out.

Prognosis.—The disease is progressive and incurable.

ACROMEGALIA.

(Marie's Disease, Pachyacria.)

Definition.—A nutritional disease, characterized by enlargement of the bones and overlying tissues, chiefly of the hands, feet, and face.

Etiology.—Unknown. It usually develops in early life. A loss of pituitary secretion is the probable cause.

Pathology.—Examination of the bones reveals a true

hypertrophy, particularly of the cancellous structures. In many cases the pituitary body has been found to be the seat of simple hypertrophy, degeneration, adenoma, or sarcoma; in a few the thymus or thyroid gland has been diseased.

Symptoms.—The hands and feet are considerably enlarged, especially in breadth; the fingers and toes are stumpy and the nails are flat and small. Hypertrophy of the inferior maxillary bone leads to elongation of the face and protrusion of the lower jaw. The lips are large and everted. Among occasional symptoms may be mentioned spinal curvature, polyuria, glycosuria, persistent headache, deafness, blindness from atrophy of the optic nerve, loss of sexual power, and in women, menstrual disorders.

Diagnosis.—Acromegalia might be mistaken for *myxœdema*, but in the latter the soft parts only are involved; the skin is firm and adherent, instead of soft and mobile as in acromegalia; and the face is round.

In *Paget's osteitis deformans* the long bones are especially involved, and are not only enlarged, but considerably deformed; and the face has a peculiar triangular shape.

Prognosis.—The affection is incurable, but the duration may be indefinite. Acute cases lasting two or three years are usually associated with sarcoma of the pituitary body.

Treatment.—So far, remedies have been futile.

SUNSTROKE.

(Heat-stroke, Thermic Fever, Coup de Soleil, Insolation, Heat-exhaustion.)

Definition.—An affection resulting from exposure to excessive heat.

Varieties.—Two varieties are observed: Thermic fever and heat-exhaustion.

Thermic Fever.

Pathology.—After death from thermic fever rigor mortis develops early and is marked. The various organs, especially the brain, are deeply congested. The left ventricle is firmly

contracted, and the right is dilated and filled with blood. The blood is dark and uncoagulated. Microscopic examination of the tissues reveals parenchymatous degeneration, or cloudy swelling.

Symptoms.—Prodromes are frequently present and consist of exhaustion, vertigo, nausea, and headache. These symptoms are followed by coma, and in this state the face is flushed; the eyes are injected; the skin is dry and burning; the temperature ranges from 106° to 112°; the pupils are contracted; the respirations are rapid and noisy; and the pulse is full and rapid. Unless the temperature soon falls the respirations become shallow, the pulse weakens, and death results in a few hours. There is a very malignant form in which the patient is suddenly stricken comatose and dies in a few hours from cardiac failure.

Sequelæ.—Meningitis; epilepsy; insanity; failure of memory; and extreme sensitiveness to high temperature.

Diagnosis.—The conditions under which the coma has developed, together with the extremely high temperature of the body, will serve to distinguish sunstroke from apoplexy, alcoholism, and uræmia.

Prognosis.—Very guarded. Probably forty per cent. perish.

Treatment.—The patient should be promptly placed in a bath of ice water and should be rubbed with ice. Ice-water enemata are also useful. Antipyrin has been administered subcutaneously with good results. When the pulse is full and strong venesection may be a valuable adjunct to the antipyretic treatment.

Heat-exhaustion.

Pathology.—According to Wood, heat-exhaustion depends on a vaso-motor paresis, as a result of which there is a determination of blood from the brain and surface of the body to the great bloodvessels of the abdomen.

Symptoms.—The mind is dazed, but consciousness is not lost; the surface is pale and cold; the skin is moist; the res-

pirations are shallow and hurried; and the pulse is rapid and feeble.

PROGNOSIS.—Recovery soon follows under appropriate treatment.

TREATMENT.—The patient should be covered with hot blankets, and hot bottles should be placed near the feet. Brandy, ammonia, and strong coffee are useful stimulants. Strychnine hypodermically is a very efficient remedy.

ALCOHOLISM.

(Dipsomania.)

Acute Alcoholism.—After excessive indulgence in alcohol the following symptoms are observed: Flushing of the face, quickening of the pulse, and mental exhilaration, followed by incoherent speech, loss of coördination, vomiting, delirium, slow pulse, subnormal temperature, and, finally, stupor and coma. Occasionally the coma is replaced or interrupted by convulsive seizures. In the majority of cases, recovery follows in the course of a day or two; but sometimes the coma deepens and death results.

Chronic Alcoholism.—This condition is characterized by a fine tremor, mental impairment, disturbed sleep, injection of the conjunctivæ, redness of the nose (acne rosacea), and the symptoms of chronic gastro-intestinal catarrh, namely, anorexia, coated tongue, fetid breath, nausea, vomiting, fulness and distress after eating, and constipation alternating with diarrhœa. When the habit is long continued, atheroma of the arteries, cirrhosis of the liver, and chronic interstitial nephritis are apt to develop.

A very common complication of chronic alcoholism is *delirium tremens* (mania a potû). This condition usually follows a protracted debauch, or spree, or is excited by an injury or some intercurrent disease. Its chief manifestations are: Mental excitement, insomnia, incoherent speech, disordered intellect, tremors, and hallucinations, usually of sight and hearing. The last are of a terrifying character; the patient hears threatening voices, or sees repulsive creatures—snakes, rats, loathsome insects, or demons—peering at him from behind

every piece of furniture. In some cases the terror excited by these hallucinations is so great that, in a fit of maniacal excitement, the patient rushes out into the street or jumps from the window. The pulse is rapid and feeble; the appetite is entirely lost; the bowels are constipated; and the temperature usually elevated (101° – 103°).

In favorable cases, in the course of a few days or a week, the excitement abates, the appetite returns, sleep is restored, and convalescence established. In unfavorable cases, typhoid symptoms are apt to develop; these are: Irregular fever, weak pulse, dry, brown tongue, stupor, subsultus tendinum, carphologia, and finally, complete coma.

Among other complications or sequelæ of dipsomania may be mentioned: Multiple neuritis, pneumonia, epilepsy, chronic meningitis, paretic dementia, and various psychoses.

Diagnosis.—The *coma of alcoholism* must be distinguished from the coma of other diseases. The history, the absence of paralysis, the subnormal temperature, the fact that the patient can be aroused by screaming in the ear, or by firm pressure over some sensitive spot like the supraorbital notch, the odor on the breath, and the absence of other cause will usually prevent an error in diagnosis.

Delirium tremens is recognized by the history, restlessness, delirium, tremors, and terrifying hallucinations.

The tremors of chronic alcoholism may be recognized by the history, the associated evidence of alcoholism, and by the fact that they are worse in the morning, and improve after the use of the stimulant.

Prognosis.—*In acute alcoholism* the prognosis should be guardedly favorable. *In delirium tremens* recovery generally follows, unless there is great debility. *In alcoholic pneumonia* the outlook is grave; recovery is exceptional. *In alcoholic neuritis* the symptoms usually subside under appropriate remedies and abstinence from the stimulant.

In *chronic alcoholism* the prognosis is generally unfavorable. When the habit is fully established, it is rarely permanently broken; temporary improvement is only too often followed by a relapse.

Treatment. *Acute Alcoholism.*—The stomach should be

emptied by the stomach-pump, a stimulating emetic, or the hypodermic injection of apomorphine (gr. $\frac{1}{10}$–$\frac{1}{8}$). If the coma persists and the pulse weakens, cardiac stimulants like ammonia, strychnia, and digitalis should be administered hypodermically. Douching and flagellation may also be employed to arouse the patient.

Delirium Tremens.—Alcohol must be withheld unless the pulse is very weak. It is essential that the patient should receive sufficient nourishment, for usually little food has been taken during the debauch which led to the delirium. Highly-seasoned beef-tea and milk with lime-water are the best foods. Sleep must be secured by chloral (gr. xx), bromide of potassium (ʒss–ʒj), hyoscine (gr. $\frac{1}{100}$), morphine (gr. $\frac{1}{4}$, and repeated once or twice), or paraldehyde (ʒj). When the pulse is weak strychnine (gr. $\frac{1}{30}$, repeated, watching the effect) is often of great value. In most cases physical restraint is essential; it is best secured by strapping the patient to the bed with sheets.

Chronic Alcoholism.—It is necessary that alcohol shall be withdrawn; the rapidity with which this can be accomplished will depend on the circumstances. In most cases the temptation to drink is so strong that confinement in an inebriate asylum is essential to the success of the treatment. Various substitutes have been recommended for alcohol, among which may be mentioned bromide of potassium, chloral, cocaine, hyoscine, and cannabis indica. As a rule, they accomplish little beyond quieting the patient and occasionally securing sleep. The diet should be nutritious, and carefully adapted to the condition of the stomach, which is usually the seat of chronic catarrh. Tonics like iron, quinine, and strychnine are often indicated. Graduated physical exercise is sometimes of decided value.

OPIUM-POISONING.

Acute Poisoning. SYMPTOMS.—A stage of excitement is followed by stupor, coma, contracted pupils, slow respirations, muscular relaxation, and a slow pulse. In the final stage the respirations become shallow and irregular, the pulse rapid and feeble, and the pupils dilated.

TREATMENT.—The stomach should be emptied by a stimulating emetic or the stomach-pump. Strong coffee may be given by the mouth. The patient should be aroused by flagellation, douching, forced walking, or the electric brush. The physiological antidotes—atropine and strychnine—should be given hypodermically in full doses, their effects being carefully watched. Electricity may be employed to stimulate respiration.

Morphine-habit. (*Morphinism, Morphiomania.*) SYMPTOMS.—Anæmia, sallow complexion, an irresistible craving for the drug, dilated pupils, tremors, loss of appetite, restlessness, insomnia, mental impairment, and a complete perversion of the moral nature.

TREATMENT.—Confinement in an asylum is nearly always necessary. The opium should be withdrawn gradually. Such substitutes as cocaine, chloral, hyoscine, paraldehyde, and sulphonal may be employed temporarily. Respiratory stimulants like strychnine, and cardiac stimulants like digitalis, are often indicated. In the vast majority of cases the habit is only suspended, not broken.

CHRONIC LEAD-POISONING.

(Plumbism, Saturnism.)

ETIOLOGY.—Chronic lead-poisoning results from the slow absorption of lead, and is most commonly observed in workmen who handle the metal. Printers, type-founders, and workers in white-lead are especially liable to be affected. Occasionally it results from the use of water which has been carried through lead pipes or which has been stored in cisterns lined with lead.

PATHOLOGY.—The muscles are degenerated, and the peripheral nerves frequently reveal evidences of chronic neuritis. In cases associated with marked muscular atrophy, poliomyelitis is discovered.

SYMPTOMS.—The following are the chief manifestations: Anæmia; severe colicky pains centering around the umbilicus and associated with retraction and rigidity of the abdominal walls; constipation; a blue line on the gums near the in-

sertion of the teeth, due to the deposition of a sulphuret of lead; paralysis; tremors; intense headache; pains in the joints (arthralgia); arterio-sclerosis; chronic interstitial nephritis; and grave cerebral symptoms (encephalopathies).

The Paralysis.—This in most instances involves the extensors of both forearms, and gives rise to the well-known wrist-drop. In advanced cases the muscles atrophy and yield the reactions of degeneration. Sensation is not affected.

Encephalopathies.—These are among the more rare manifestations of plumbism, and consist of convulsions, coma, delirium, intense headache, and blindness from atrophy of the optic nerves.

Prognosis.—Guardedly favorable.

Treatment.—Prophylaxis consists in absolute cleanliness; the use of respirators in lead factories; the avoidance of eating in an atmosphere laden with the dust of the metal; and in the occasional use of Epsom salts.

The curative treatment consists in the administration of iodide of potassium (gr. v–x thrice daily) and the use of sulphur baths. Constipation should be relieved by Epsom salts. The colic may require the hypodermic injection of morphine and atropine, and the application of hot fomentations to the abdomen. The paralysis generally yields to massage, the constant current, and hypodermic injections of strychnine.

CHRONIC MERCURIAL POISONING.

Etiology—This is usually observed in those employed in quicksilver mines, or engaged in making mirrors, barometers, or other scientific instruments requiring the use of mercury.

Symptoms.—Anæmia, loss of flesh and strength, gastro-intestinal disturbances, and marked tremors. The latter usually begin in the extremities, and are at first slight, but later the whole body is involved, and the tremors are violent. In advanced cases they may continue during sleep. Grave cerebral symptoms occasionally develop, such as vertigo, headache, impairment of intellect, convulsions, paralysis, and coma.

Diagnosis.—The history, the marked tremor of the head,

and the absence of the peculiar gait (festination) will distinguish it from *paralysis agitans.*

The history and the absence of nystagmus will distinguish it from *disseminated sclerosis.*

TREATMENT.—Removal from the influence of the metal. Tonics. Iodide of potassium. Electricity. Sedatives for the tremors.

CHRONIC ARSENICAL POISONING.

ETIOLOGY.—It is observed in workmen employed in arsenic works and glass factories. Inhaling the dust of fabrics, papers, artificial flowers etc., which have been colored with arsenic, may induce poisoning.

SYMPTOMS.—Anæmia, loss of flesh and strength, conjunctivitis, gastro-intestinal catarrh, loss of hair, cutaneous eruptions, and paralysis. The last, unlike that observed in lead-poisoning, usually involves the extensors of the legs, but later it may also involve the arms.

TREATMENT.—Removal from the influence of arsenic. Tonics. Electricity and massage to the affected muscles.

DISEASES

OF THE

SKIN AND ITS APPENDAGES.

THE COLOR OF THE SKIN.

Pallor as a *permanent condition* is generally an expression of anæmia; but it should be borne in mind that in some cases the surface is pale when the blood is normally rich in corpuscles and hæmoglobin; and that in other cases the surface has a natural color when the blood is considerably deficient in corpuscles and hæmoglobin. It follows therefore that an absolute diagnosis of anæmia must rest on an analysis of the blood.

Pallor as a *temporary condition* may result from emotional excitement, exposure to extreme cold, shock, syncope, or collapse.

Yellowness of the skin may result from *jaundice*, in which case the conjunctivæ will also be yellow and the urine will contain bile. Yellowness may also result from *chlorosis or pernicious anæmia*, and in these cases the normal color of the conjunctivæ, the associated symptoms of the disease, and the absence of bile in the urine will indicate the cause.

Whiteness of the Skin.—A milk-white hue over extensive areas may be observed in *albinism*, *vitiligo*, and in *leprosy*.

Dark-brown or gray discoloration of the skin is observed in the following conditions:—

Addison's Disease.—In this affection the skin has a bronzed appearance, which is especially marked on exposed parts; the

buccal mucous membrane may also reveal discolored plaques; and there are in addition anæmia, prostration, and gastric irritability.

Argyria.—This term is applied to the dark-gray discoloration of the exposed parts which follows the prolonged use of nitrate of silver. The discoloration is due to a deposition of the oxide of silver, and is more or less permanent. It is said to be preceded by a dark line on the gums, similar to the one observed in chronic lead-poisoning. Formerly, when nitrate of silver was used extensively in the treatment of epilepsy, it was not an uncommon condition.

Vagabondismus.—This term is applied to the dark-brown discoloration of the skin which follows prolonged exposure to the weather, uncleanliness, and perhaps the irritation of the skin resulting from pediculosis.

Blueness of the skin, as a permanent condition, is generally an expression of cyanosis.

Hardness, or Induration of the Skin.

Induration of the skin is observed in *scleroderma.* In this affection the skin is tense, hide-bound, and more or less pigmented. Induration is also observed in *myxœdema.* In this condition the skin is swollen as in œdema, but it is firm, inelastic, and does not pit on pressure. In addition, the features are peculiarly broadened and the mental power is impaired. Circumscribed patches of induration are observed in *morphœa.* The circumscribed patches, with hyperæmic or pigmented borders, and the smooth, shiny, atrophied skin are the diagnostic features.

Œdema, or *dropsy of the subcutaneous tissues,* when extreme, also causes induration.

A brawny, indurated condition of the muscles, especially of the legs, is frequently observed in *scurvy.* It probably results from a sanguineous exudation. The anæmia, purpuric spots, and spongy, bleeding gums will aid in the diagnosis.

ŒDEMA, OR DROPSY OF THE SUBCUTANEOUS TISSUES.

Œdema may be recognized by a swelling which pits on pressure. It results from : (1) Venous stasis—from chronic heart, liver, and lung disease; and from local obstruction to the venous circulation, as by a tumor, pregnant uterus, or a varicose condition of the veins. (2) Alterations in the blood or capillaries, as in Bright's disease, anæmia, and inflammation.

GLOSSY SKIN.

"Glossy Skin."—This term was applied by Paget to indicate a smooth, atrophied, and shiny appearance of the skin. It is most frequently observed after inflammation or injury of the nerve-trunks. It is sometimes associated with an intense burning pain, to which Mitchell has given the name *causalgia*,

ENLARGEMENT OF THE SUPERFICIAL VEINS.

Enlargement of the superficial veins may result from chronic heart, lung, or liver disease; from the pressure of a tumor or aneurism on deep-seated veins; or, as a general condition, it may be congenital and result from occlusion of deep veins.

"Caput Medusæ."—This term is applied to a circle of dilated veins surrounding the umbilicus. It is indicative of obstruction to the portal circulation, and may result from atrophic cirrhosis of the liver, from thrombosis of the portal vein, or from the pressure of a tumor on the portal vein.

CUTANEOUS EMPHYSEMA.

Cutaneous emphysema consists in an escape of air into the cellular tissue. It is manifested by a diffuse, pallid swelling of the skin, which crackles on palpation and which pits on pressure; but, unlike œdema, the depression immediately disappears when the finger is withdrawn. It may result (1) from

traumatism of the air-passages, as a gunshot wound of the chest or a fracture of the rib. (2) From rupture of the œsophagus, stomach, intestines, larynx, trachea, or lungs. The rupture of these organs is usually due to ulceration, as in cancer of the œsophagus, tuberculous cavity of the lung, or purulent pleurisy; but occasionally the lung ruptures from violent strain.

ABNORMAL CONDITIONS OF THE NAILS.

Atrophy of the Nails.—The nails may become dry, brittle, discolored, and cracked in organic disease of the spinal cord; after inflammation or injury of the peripheral nerves; after prolonged febrile diseases, like typhoid fever; and in certain affections of the skin which involve the matrix of the nail, as eczema, psoriasis, and ringworm.

Curving of the Nails.—Incurvation of the nails is generally associated with clubbing of the terminal phalanges. It is observed in phthisis, chronic cardiac disease, and in many wasting diseases.

Onychia.—Inflammation of the matrix of the nail may result from injury; from syphilis; from organic disease of the spinal cord, as locomotor ataxia; from arthritis deformans; and from cutaneous affections involving the matrix, as leprosy, ringworm, and eczema.

CUTANEOUS ERUPTIONS.

Macules.

Macules are discolored spots which are neither elevated nor depressed.

A general red macular eruption is observed in the following conditions:—

Syphilis.—Secondary syphilis may manifest itself as an eruption of small red macules. They are usually abundant and frequently cover the entire body; they lack subjective symptoms; they are usually associated with the history or with the evidences of syphilis, such as the scar of the chancre, bone-pains, alopecia, swollen glands, and sore throat.

Erythema Multiforme may manifest itself as a macular eruption, but the macules are usually associated with dark-red papules or tubercles. The multiformity of the lesions; their preference for the extremities; their appearance in successive crops; the short duration of each lesion; the absence of subjective phenomena, such as itching and burning; and the presence of rheumatic pains are the diagnostic features.

Pityriasis rosea.—The eruption is especially found on the trunk; the lesions are rose-red in color; they are slightly scaly, the scales being dry; subjective phenomena are generally absent; and the duration is a few weeks.

Pediculosis Corporis.—Lice may produce a minute red or purple eruption. The small size of the lesions; their confinement to the covered parts; the intense itching and the presence of scratch-marks; and the discovery of pediculi on the clothes are the diagnostic features.

Rötheln.—This affection produces a macular or maculopapular rash which disappears in two or three days by slight desquamation. The moderate fever, sore throat, swollen cervical glands, and history of contagion will assist in the diagnosis.

Accidental Rashes.—Local inflammation like tonsillitis and acute gastritis, and certain drugs and foods occasionally produce a macular rash.

Purpuric spots, or hemorrhagic macules (petechiæ), result from minute extravasation of blood into the skin.

A purpuric eruption is observed in the following conditions:—

Purpura Hæmorrhagica (*Morbus Maculosus Werlhofii*).—This affection occurs especially in children; it is associated with fever and bleeding from the mucous membranes; and generally runs a course of one or two weeks.

Scurvy.—This disease results from a deprivation of fresh vegetables, and is associated with spongy, bleeding gums, great weakness, and a brawny induration of the muscles.

Rheumatism.—Occasionally an eruption of purpuric spots appears in rheumatic subjects. It is usually associated with pains in the limbs, but fever is generally absent.

Peliosis Rheumatica (*Schönlein's Disease*).—This is an acute affection characterized by purpuric spots, urticaria, sore throat, moderate fever, and an inflammation of the joints resembling rheumatism. By some the disease is regarded as a manifestation of rheumatism.

Extreme Anæmia.—A petechial rash is not uncommon in pernicious anæmia, leucocythæmia, cancer, and advanced Bright's disease. The history and the associated symptoms of the original disease will indicate the diagnosis.

Certain Infectious Diseases.—In typhus fever a purpuric eruption appears on the fourth or fifth day. In cerebro-spinal meningitis the eruption is frequently petechial. In malignant measles and malignant smallpox the rash is often hemorrhagic. In acute yellow atrophy of the liver and in ulcerative endocarditis a petechial eruption is frequently observed.

Poisoning from Certain Substances.—Poisoning from phosphorus, the virus of venomous snakes, mercury, and antipyrin may be associated with an eruption of purpura.

Pediculosis and Kindred Affections.—Body-lice, bed-bugs, and fleas produce petechial lesions which are surrounded by slight areolæ. The itching, scratch-marks, and discovery of the parasite are the diagnostic features.

Brown macules are observed in :—

Lentigo, or Freckle.—The spots are small, and are fonnd especially on exposed parts—face, neck, shoulders, and hands.

Chloasma.—Dark spots may result from irritation of the skin from the action of chemicals, heat, scratches, or blisters. They are sometimes noted in general diseases like Addison's disease and syphilis. They also occur in primary affections of the skin, as vitiligo, morphœa, scleroderma, and leprosy.

Moles, or Nævus Pigmentosa.—These consist in congenital deposits of pigment on various parts of the body.

White or pale yellow macules are observed in :—

Vitiligo.—Apart from the absence of pigment, the skin is normal in appearance and function. An excess of pigment is generally noted at the periphery of the white patches.

Leprosy.—In this condition there are structural changes in the skin and anæsthesia in addition to the white appearance.

Morphœa.—In the late stage of this affection the circumscribed patches are white or yellow. The structure of the skin is altered, and the periphery of the patches is distinctly hyperæmic.

Facial Hemiatrophy.—The onset of this disease may be marked by the appearance of a yellow or white spot on one side of the face.

Diffuse Erythema or Inflammation of the Skin.

Diffuse erythema or inflammation of the skin may result from :—

The Action of Certain Drugs (*Dermatitis Medicamentosa*).—Belladonna, quinine, chloral, cubebs, salicylic acid, and arsenic may produce a diffuse red rash.

Scarlet Fever.—The history of contagion, high fever, sore throat, swollen glands, rapid pulse, and the punctiform character of the rash will indicate the diagnosis.

Rötheln.—In some cases of rötheln the eruption is red and diffuse. The history, slight fever, slight catarrh, and marked swelling of the post-cervical glands will suggest rötheln.

Local irritation from traumatism, excessive heat, poisonous plants or drugs.

Erythema Intertrigo.—This occurs where two cutaneous surfaces come in contact. The part is red, moist, and sometimes macerated. The condition excites a burning pain.

Eczema.—The skin is thickened and infiltrated; there is marked itching; the redness shades off gradually; and there is no fever.

Erysipelas.—The part is considerably swollen; the redness and swelling terminate in an abrupt ridge; and the temperature is high.

Acne Rosacea.—This is a chronic disease; the redness appears on the face, and is associated with acne lesions and dilated capillaries.

Vesicles.

A vesicle is a small elevation of the skin, containing serous fluid, and varying in size from a pinhead to a split-pea. Vesicles are observed in the following conditions :—

Sudamen.—This consists of an eruption of minute vesicles which result from the imprisonment of sweat in the layers of the skin. It is usually associated with free perspiration; the vesicles are translucent, lack inflammatory characteristics, and show no tendency to rupture.

Herpes.—The vesicles appear in groups or clusters; they are mounted on an inflammatory base; they show no tendency to rupture; they are frequently associated with burning or neuralgic pains; and they are distributed along the line of the nerve-trunks.

Dermatitis Venanata.—A vesicular eruption may result from contact with poisonous plants, such as the poison ivy or oak. The eruption generally appears on the exposed parts—face or hands; the part is red and swollen and there is intense itching.

Dermatitis Herpetiformis.—The vesicles are very irregular in shape; they appear in clusters; they are very tense; they show no tendency to rupture; they are frequently associated with other lesions—papules, pustules, and bullæ; they excite intense itching; and they appear in crops over a period of weeks or months.

Impetigo Contagiosa.—The eruption consists of small vesicles which subsequently enlarge until they reach the size of blebs; the vesicles appear in crops; are commonly discrete; are flat and umbilicated; are filled with a straw-colored fluid; they show no tendency to break, but dry up and form thin yellow crusts, and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from one to two weeks.

Vesicular Eczema.—The vesicles are quite small and are aggregated in patches; the intervening skin is red and thickened; the vesicles tend to break and pour forth a serous fluid which keeps the part moist; and the eruption is associated with intense itching.

Miliaria, or Heat-rash.—This may appear as an eruption of minute vesicles; they are alway discrete; they are surrounded by red areolæ; they usually appear on the trunk; they are generally associated with pin-head papules; they

show no tendency to rupture; and they excite a little burning and itching.

Scabies.—In this affection the vesicles are small; they are usually associated with pustules and *burrows;* they excite intense itching; and they are usually found on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs.

Blebs, or Bullæ.

A bleb, or bulla, is a circumscribed elevation of the skin, containing serous fluid, and varying in size from a pea to an egg. Blebs are observed in the following conditions:—

Impetigo Contagiosa.—The blebs are flat and umbilicated; they contain a straw-colored fluid; they appear in crops; they are commonly discrete; they show no tendency to break, but dry up and form thin yellow crusts; and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from one to two weeks.

Dermatitis Herpetiformis.—The bullæ are frequently associated with papules, vesicles, and pustules; they are surrounded by inflamed skin; they appear in clusters; they show no tendency to break, but dry up and leave yellowish-brown crusts; and they excite considerable itching.

Pemphigus.—The bullæ appear in crops; excite but little itching; they lack an inflammatory areola; and as a rule they dry up, and leave behind a thin pellicle. The disease is generally chronic.

Syphilis.—The bullous syphilide is observed in hereditary syphilis, and very late in the acquired disease. The contents of the bullæ soon become pustular; the blebs dry up, and form dark-green, cone-shaped, stratified crusts, which become detached and leave discharging ulcers. The history and the other evidences of syphilis will aid in the diagnosis.

Pustules.

A pustule is a small circumscribed elevation of the skin containing pus. Pustules are observed in the following diseases:—

Eczema Pustulosum.—The pustules are small; are aggregated in a patch; are generally associated with minute vesicles; the intervening skin is red and thickened; and there are marked burning and itching.

Acne Vulgaris.—The pustules are usually confined to the face, back, and shoulders; they have their origin in the sebaceous follicles; they are generally associated with papules and comedones; and they excite no itching.

Dermatitis Herpetiformis.—The pustules are frequently associated with papules and vesicles; they are surrounded by inflamed skin; they appear in clusters; and they excite considerable itching.

Impetigo Simplex.—This affection is usually observed in children; the pustules are round, and range in size from a pea to a cherry; there is only a slight red areola, and this finally disappears; the pustules remain discrete; they show little tendency to rupture, but dry up and form yellowish-brown crusts; they are mostly observed on the extremities; they excite no itching. The disease lasts from a few days to a week.

Impetigo Contagiosa.—The eruption is at first vesicular, but it soon becomes pustular; the pustules vary in size from a pea to a large marble; they are flat and umbilicated; they appear in crops; they are commonly discrete; they show no tendency to break, but dry up and form thin yellow crusts; and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from one to two weeks.

Varicella, or Chicken-pox.—The pustules result from vesicles; they appear especially on the trunk; they are small and not umbilicated; they excite but little itching. There is some fever. The disease lasts but three or four days.

Ecthyma.—This disease is observed especially in poorly-nourished adults. The pustules vary in size from a pea to a cherry; they are few in number; they are mounted on an inflammatory base, and are surrounded by a distinct inflammatory areola; they excite but little itching; they seldom break, but dry up and form brownish crusts.

Smallpox.—In this disease shot-like papules and umbilicated vesicles precede or are associated with the pustules. The latter are small, surrounded by a red areola, and usually excite some itching. The high fever and history of contagion will assist in making the diagnosis.

Syphilis.—The pustules are frequently associated with other lesions; they are often mounted on a copper-colored inflammatory base; they excite no itching; and they are usually associated with the history and the other evidences of syphilis.

Scabies.—The pustules are small and usually associated with papules, vesicles, and *burrows;* they are especially observed on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs, and they excite considerable itching. There is often a history of contagion.

Papules.

A papule is a circumscribed solid elevation of the skin varying in size from a pin-head to a pea. Papules are observed in the following conditions:—

Erythema Multiforme.—The papules are often associated with macules and tubercles; they are flat, and are of a bright-red or purple color; they appear especially on the extremities; and they show no tendency to suppurate, but gradually disappear in the course of two or three weeks; they excite no itching, but they are often associated with prostration and rheumatic pains.

After the Use of Certain Drugs.—Bromides, iodides, copaiba, cubebs, and tar may produce a papular eruption. The history will aid in the diagnosis.

Eczema Papulosum.—The papules are very small, closely aggregated, and often associated with vesicles and pustules; the skin is thickened; and there is intense itching.

Miliaria, or Prickly Heat.—The papules are very small; they are very often associated with minute vesicles; they always remain discrete; they appear especially on the trunk; and they excite a little burning and itching.

Acne Vulgaris.—The papules are usually confined to the face, back, and shoulders; they are generally associated with

pustules and comedones; they involve the sebaceous follicles; and they do not excite subjective symptoms.

Scabies.—The papules are small and are usually associated with pustules, vesicles, and *burrows;* they are especially observed on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs; and they excite considerable itching. There is often a history of contagion.

Syphilis.—The papules are dark in color; they are widely distributed, being especially marked on the trunk and flexor surfaces of the extremities; they are usually associated with pustules; and they excite no itching. The history and the accompanying evidences of syphilis will aid materially in establishing the diagnosis.

Smallpox.—The papules are hard and have a shot-like feel; they soon terminate in umbilicated vesicles; they excite some itching, and they are associated with high fever. pain in the back, and often a history of contagion.

Measles.—The papules are small, and run together to form crescentic-shaped patches; and they are associated with moderate fever, swollen cervical glands, coryza, conjunctivitis, and bronchitis. There is often a history of contagion.

Tubercles.

Tubercles are large, circumscribed, solid elevations of the skin varying in size from a large pea to a walnut. They are observed in the following conditions:—

Erythema Nodosum.—The tubercles are large; they usually appear on the extremities; they are reddish-purple in color; they never suppurate; and they are associated with malaise, fever, and rheumatic pains.

Erythema Multiforme.—The tubercles are generally associated with macules and papules; they are flat, and are of a bright-red or purple color; they appear especially on the extremities, and they show no tendency to suppurate, but gradually disappear in the course of two or three weeks. They excite no itching, but are often associated with prostration and rheumatic pains. The disease is probably allied to erythema nodosum.

Lupus Vulgaris.—This may begin as a papule or tubercle. It is especially observed on the face. The tubercles are of a pale-red color and are quite soft to the touch. As a rule, they slowly break down and form shallow ulcers with soft red margins. The ulcers are painless and secrete but little material. They may invade all of the soft structures, but the bones escape.

Syphilis.—The tubercular syphilide manifests itself as dark-red tubercles. There are seldom more than three or four, and they generally appear on the face and extremities. They are very firm, and often break down, forming deep, punched-out ulcers which secrete an abundant purulent material.

Tinea Sycosis, or Barber's Itch.—The tubercles appear on the hairy parts of the face and involve the hair-follicles. Suppuration soon begins in the centre of the tubercles, and the hairs become dry, brittle, and loose. The microscope will reveal the tricophyton.

Leprosy.—One form of leprosy manifests itself as tubercles. The latter are of a pale-red or yellow color, and undergo slow absorption or ulceration. There is usually more or less anæsthesia in the parts affected.

Wheals, or Pomphi.

Wheals are evanescent elevations of the skin, generally more or less round, and often white in the centre and pale-red at the periphery. They excite considerable itching. They are observed in the following conditions:—

Urticaria.—The wheals appear in crops; they are of very short duration; they may appear on any part of the body; and they excite intense itching.

Erythema multiforme, *peliosis rheumatica* (Schönlein's disease), and certain insects like mosquitoes also produce wheals.

Crusts.

Crusts consist in dried exudation, and may be red, yellow, brown, or green in color. They are marked in the following diseases:—

Eczema.—The crusts are generally associated with pustules and vesicles; the surrounding skin is red and thickened; and there is considerable itching.

Seborrhœa.—Crusts of seborrhoea are generally observed on the scalp. Itching is absent, and there are no evidences of inflammation.

Syphilis.—The crusts are thick; they are of a dark-brown or green color; and they are often associated with ulcers which freely discharge. The history and other evidences of syphilis will aid in the diagnosis.

Impetigo.—The crusts are thin and yellow; and they are associated with blebs which appear in crops.

Favus.—The crusts generally appear on the scalp; they are yellow, brittle, and cup-shaped; they are usually perforated by a hair, and have a peculiar musty odor.

Tinea Tonsurans, or Ringworm of the Scalp.—In neglected cases this affection may be associated with crusting. It is only observed in children. The grayish scales, the dry, brittle, and broken hairs projecting through the crusts, the alopecia, and the detection of the tricophyton are the diagnostic features.

Scales.

Scales are dry exfoliations from the upper layers of the skin. They are observed in the following diseases:—

Squamous Eczema.—The scales are usually associated with papules; the underlying skin is red and thickened; and there is often marked itching.

Seborrhœa Sicca.—The scales are greasy, and the underlying skin shows no evidence of inflammation. The sebaceous follicles are often dilated.

Psoriasis.—The scales are dry, and are of a pearly-white color; they are associated with circumscribed, sharply-defined, elevated inflammatory patches. The extensor surfaces are especially involved. There is little or no itching.

Ichthyosis.—This affection begins in early life. The scales are dry, and are especially marked on the extensor surfaces. Itching is absent, and there is no evidence of inflammation.

Syphilis.—The scales are dry, and are of a grayish color; they are usually associated with papules; and they are especially marked on the palms and soles. There is no itching. The history and other evidences of syphilis will assist in the diagnosis.

Pityriasis Rosea.—The scales are found especially on the trunk, and are associated with small, rose-red macules. There is no itching. The disease runs an acute course of a few weeks' duration.

Ringworm.—The scales are dry and scant; they are associated with circumscribed red patches which tend to disappear in the centre. There is often marked itching. Microscopic examination reveals the tricophyton.

Ulcers.

Ulcers are observed especially in the following diseases:—

Syphilis.—The ulcers are deep; they have a punched-out appearance; they secrete an abundant offensive material; they often involve the bone; they extend rapidly; they are not painful, and the imperfect cicatrix which they produce is soft. The history and other evidences of syphilis will aid in the diagnosis.

Epithelioma.—This appears in late life; there is usually a single centre of ulceration; the ulcer is irregular in shape; the edges are thickened and infiltrated; the secretion is scanty and bloody; the progress is somewhat slow, and there is often pain.

Lupus Vulgaris.—This generally appears in early life; there are often several centres of ulceration; the ulcers are usually superficial; the edges are not thickened; the progress is extremely slow; the bones are never involved; there is very little secretion, and soft papules often develop in the cicatrix, which is firm and contracted.

Simple Ulcers may result from traumatism, the application of caustics, or the action of intense heat or cold. Ulcers are frequently observed on the legs of old people in association with varicose veins. Simple ulcers may be recognized by the history, location, appearance, and the absence of other causes.

Perforating Ulcer of the Foot.—This term is applied to a deep-seated ulcer appearing on the sole of the foot and most frequently observed in locomotor ataxia. It usually begins as a corn in the neighborhood of the great toe, and is generally associated with anæsthesia of the sole of the foot.

Decubitus.—This term is applied to the bedsores which form after the occurrence of grave cerebral or spinal lesions. They are generally observed on parts which are subjected to pressure, as the sacrum, buttocks, calves, and heels, and are preceded by erythema and vesication.

DISEASES OF THE SWEAT-GLANDS.

Anidrosis.

DEFINITION.—A deficiency of sweat.

ETIOLOGY.—It may be a symptom of some general disease, like diabetes or Bright's disease; it may be an associated condition in certain cutaneous diseases, such as ichthyosis or psoriasis; and it may develop without obvious exciting cause as a result of disturbed innervation.

TREATMENT.—Remedies should be directed to the primary disease.

Hyperidrosis.

DEFINITION.—Excessive sweating.

ETIOLOGY.—As a general condition it is often observed in phthisis and in other diseases characterized by marked debility. Local hyperidrosis is most frequently observed in the hands, feet, and axillæ, and probably results from some derangement of the sympathetic nervous system. Unilateral sweating of the face may indicate an aneurism or tumor pressing on the cervical sympathetic.

SYMPTOMS.—The primary symptom is excessive sweating, and this often leads to intertrigo or eczema. Bromidrosis is often associated with the hyperidrosis.

PROGNOSIS.—Guarded. In many cases the condition is very obstinate.

TREATMENT.—Frequently there is an evident impairment of the general health which will require appropriate treatment. Internally, one of the following remedies may be employed to diminish the amount of sweat: Belladonna, picrotoxin, agaricin, or ergot.

Local Treatment.—Dusting-powders of starch, talc, or lycopodium with boric or salicylic acid; or lotions containing sulphate of zinc, tannic acid, or alum, are often very useful.

℞ Pulv. acid. salicylic.,
Pulv. zinci carb. præcip.,
Pulv. magnesii ustæ, āā ʒiv;
Pulv. amyli, ʒxv;
Pulv. talci, ʒxx.—M. (HARDAWAY.)

Sig.—Dusting-powder.

In hyperidrosis of the feet the method suggested by Hebra is often very efficient. The feet should be washed, thoroughly dried, and then carefully enveloped in strips of muslin which have been spread with diachylon ointment. The application should be made twice daily. In the dressing no water should be employed, but the feet must be carefully wiped and then dusted with starch or lycopodium before the ointment is re-applied. The treatment should be continued for from one to two weeks, after which the feet may be washed and the dusting-powder alone used

Bromidrosis.

(Osmidrosis.)

DEFINITION.—A functional affection characterized by the excretion of sweat which has a fetid odor.

SYMPTOMS.—It is generally local and often confined to the feet; it is frequently associated with hyperidrosis.

TREATMENT.—Same as hyperidrosis.

Chromidrosis.

DEFINITION.—A functional affection characterized by the secretion of colored sweat.

SYMPTOMS.—The parts most frequently affected are the face and trunk; the most common colors are red and yellow. It is often associated with hyperidrosis.

Sudamen.

DEFINITION.—A cutaneous affection characterized by the eruption of minute vesicles resulting from the retention of sweat in the layers of the skin.

ETIOLOGY.—It is often observed in health in persons who perspire freely. It is frequently noted in febrile diseases which are associated with sweating, like pneumonia and typhoid fever.

SYMPTOMS.—Minute, irregular, translucent vesicles appear on the surface. They are not surrounded by an inflammatory

areola. They do not rupture, but dry up and are followed by slight desquamation.

TREATMENT.—The affection has little significance and treatment is rarely required.

FUNCTIONAL DISEASES OF THE SEBACEOUS GLANDS.

Seborrhœa.

(Steorrhœa.)

DEFINITION.—A functional affection characterized by excessive secretion of sebaceous material which may be normal or perverted.

ETIOLOGY.—In many cases the cause is not apparent. Often the disease is associated with impairment of the general health. By some it is regarded as of parasitic origin.

VARIETIES.—Seborrhœa sicca and seborrhœa oleosa.

Seborrhœa Sicca.—This form is most frequently observed on the scalp and constitutes what is popularly termed *dandruff*. Examination reveals an incrustation composed of thin, yellowish-gray, greasy scales. In uncomplicated cases the skin is pale, but from irritation it may subsequently become hyperæmic or inflamed. When allowed to continue, the nutrition of the hair is interfered with and baldness results.

On the body seborrhœa sicca appears as yellowish-gray slightly elevated patches covered with greasy scales. The outlets of the follicles are often dilated. There is generally more or less redness of the skin from hyperæmia (*seborrhœal eczema.*)

Seborrhœa Oleosa.—This form is most commonly observed on the face, particularly about the nose, which is habitually bathed in an oleaginous material which has exuded from the sebaceous follicles. From irritation the parts are often red. The condition is frequently associated with seborrhœa sicca, comedo, and acne.

DIAGNOSIS. *Eczema.*—In this disease the skin is red and thickened; there is marked itching; and the scales are not greasy.

Psoriasis.—In this disease the scales are dry and pearly and there are evidences of inflammation.

PROGNOSIS.—Favorable under prolonged and judicious treatment.

TREATMENT—The general health may be impaired; hence tonics like iron, strychnine, and cod-liver oil are often indicated. The gastro-intestinal tract will often require especial attention. Constipation should be relieved by diet, enemata, or mild laxatives.

Local Treatment.—Crusts should be removed by applications of oil, followed by shampooing with alcohol and green soap. When the scalp is thoroughly clean, one of the following remedies may be applied: Sulphur, mercury, salicylic acid, carbolic acid, or resorcin.

℞ Ceræ alb., ʒij;
Petrolat. liquid., fʒij;
Aquæ rosæ, fʒvij;
Sodii borat., gr. x;
Sulphur., ʒij.—M.

Fiat unguentum.
Sig.—Apply at bedtime for several nights, then shampoo.

Or—

℞ Acid. carbolic., ♏xxx;
Olei ricini, fʒij;
Alcoholis, fʒj–ʒvj.—M.
(DUHRING and STELWAGON.)

Sig.—Fill an eye-dropper, introduce between the hairs, and subsequently rub in by means of a flannel rag.

Mild cases of facial seborrhœa often yield to the following ointment:—

℞ Hydrarg. chlor. mit., gr. xx;
Ung. zinc. oxid., ʒj.—M.

Sig.—Apply at bedtime.

Comedo.

DEFINITION.—A functional disease of the sebaceous glands, characterized by the retention of discolored sebaceous material in the distended ducts of the gland.

ETIOLOGY.—It is most frequently observed in young adults. Debility, gastro-intestinal disorders, anæmia, and lack of cleanliness are predisposing factors.

PATHOLOGY.—The material in the ducts is composed of sebum, altered epithelium, and pigment matter which is probably derived from without. Microscopic examination of the material often reveals a mite—the *demodex folliculorum*—but its presence is accidental and of no etiological significance. Comedo is generally associated with seborrhœa.

SYMPTOMS.—The disease is characterized by an aggregation of minute black or yellowish spots which correspond to the outlets of the sebaceous glands. The lesion is often slightly elevated, and when the skin is squeezed a white filiform mass exudes, to which the term "flesh-worm" has been popularly applied. The parts most commonly affected are the face, back, and ears. The condition frequently excites an inflammation of the follicles, hence it is often associated with acne.

PROGNOSIS. — Favorable under persistent and judicious treatment.

TREATMENT.—Anæmia, dyspepsia, and constipation must be treated by a careful regulation of the personal hygiene, and by the use of appropriate remedies. Tonics like iron, quinine, cod-liver oil, and strychnine are often indicated.

Local Treatment. — Large plugs may be pressed out by means of a watch-key or a special instrument for the purpose. Softening and removal of smaller plugs may be hastened by the application of cloths wrung out in very hot water. Kneeding and the application of alcohol and green soap will also assist in their expulsion. Mercury and sulphur are useful remedies.

℞ Hydrarg. chlor. corros., gr. iv;
Alcoholis, f℥j;
Aquæ rosæ, q. s. ad f℥iv.—M.
Sig.—Dab on twice daily.

Milium.

(Grutum.)

DEFINITION.—An affection characterized by the appearance of small, pearly, non-inflammatory elevations, which result from the accumulation of inspissated sebum in ducts, the outlets of which have been occluded.

Symptoms.—It is generally observed about the face, and consists of a collection of small, round, pearly elevations, which vary in size from a pin-head to a millet-seed. The contents of the distended duct cannot be squeezed out until an opening is made, and thus it differs from comedo. It is frequently associated with comedo and acne.

Treatment.—Incise the lesion, express the contents, and touch with tincture of iodine.

Steatoma.

(Wen.)

Definition.—A steatoma, or wen, is a cyst resulting from the retention of secretion in a sebaceous gland.

Symptoms.—One or more rounded or oval elevations, varying in size from a pea to a large walnut, slowly appear on the scalp, face, or back. They are painless, rather soft, and when opened are found to contain a yellowish-white caseous mass.

Diagnosis. *Fatty Tumors.*—Fatty tumors are rare on the scalp; they are frequently lobulated; they have a doughy feel; and are not so movable as wens.

Treatment.—The sack and its contents should be carefully dissected out. Simple excision and evacuation are always followed by a return of the cyst.

ERYTHEMA SIMPLEX.

Definition.—Active hyperæmia of the skin.

Etiology.—It may result from exposure to heat or cold; from traumatism; or from the application of some irritating substance. A symptomatic variety is frequently observed in gastric irritation and systemic diseases.

Symptoms.—Diffuse uniform redness, disappearing on pressure, and without thickening or elevation of the skin. When it is marked, there may be slight burning.

Treatment.—Sedative lotions or dusting-powders.

ERYTHEMA INTERTRIGO.

(Chafing.)

DEFINITION.—Hyperæmia induced by the attrition of opposing surfaces of the skin.

ETIOLOGY.—It is common in children and in fat subjects. It is especially noted where there are friction and perspiration, as under pendulous mammæ, between the upper parts of the thighs, and around the genitalia.

SYMPTOMS.—It is characterized by diffuse redness, and often by heat and moisture. It excites a burning sensation. When the cause is continued it may result in dermatitis.

TREATMENT.—Apply a lotion of boric acid and follow with a dusting-powder.

ERYTHEMA NODOSUM.

(Dermatitis Contusiformis.)

DEFINITION.—An acute inflammatory disease, characterized by crops of large bright-red nodes which in the process of evolution assume different colors as in the fading of a bruise.

ETIOLOGY.—It is usually seen in children. It is frequently associated with rheumatic and digestive disturbances.

SYMPTOMS.—There is a sudden eruption of bright-red nodes varying in size from a pea to an egg. The extremities are most commonly affected. The advent is marked by malaise, headache, slight fever, and rheumatoid pains. At first the lesions resemble boils, but unlike the latter, they do not suppurate, but gradually turn yellow, blue, and green as a bruise.

PROGNOSIS.—Favorable. Duration a few weeks.

TREATMENT.—Saline laxatives and sodium salicylate are recommended. Locally, lead-water and laudanum make a soothing application.

ERYTHEMA MULTIFORME.

DEFINITION.—An inflammatory disease characterized by erythematous, papular, vesicular, or bullous lesions.

Etiology.—It is more common in women than in men. It is apt to develop in the spring or fall. Rheumatism and gastro-intestinal disturbances seem to predispose.

Symptoms.—It is marked by an eruption, usually on the extremities, of the following lesions: macules, papules, vesicles, or bullæ. The lesions may aggregate or remain discrete; they last one or two weeks and gradually fade. There is little or no itching. In some cases there is decided constitutional disturbance, manifested by malaise, headache, slight fever, and rheumatic pains.

Diagnosis. *Dermatitis Herpetiformis.* — The marked itching, the greater tendency for the lesions to cluster, and the chronic character of dermatitis herpetiformis will usually prevent an error in diagnosis.

Urticaria.—In this disease the individual lesions last a very short time and are associated with marked itching.

Prognosis.—Favorable. Duration a few weeks.

Treatment.—In the debilitated iron and quinine are useful. In the rheumatic, the salts of lithium and of potassium may be employed. Constipation should be relieved by saline laxatives. Locally, lotions of boric or carbolic acid followed by dusting-powders exert a beneficial effect.

URTICARIA.

(Hives, Nettle Rash.)

Definition.—An inflammatory affection characterized by the eruption of pale-red, evanescent wheals which are associated with severe itching.

Etiology.— Gastro-intestinal disturbances, emotional excitement, and chronic visceral diseases predispose. In some it may be excited by certain articles of food such as shellfish, strawberries, etc. The bites of certain insects produce the disease, such as mosquitoes, bed-bugs, and caterpillars. Some drugs induce urticaria in susceptible people.

Pathology.—The disease consists in a vaso-motor spasm, followed by paresis of the vessels and an outpouring of serum.

Symptoms.—There is a sudden general eruption of papules or wheals which is associated with intense itching. Each

lesion lasts a few hours and is succeeded by new ones in other places.

VARIETIES. *Urticaria Papulosa.*—In this form the wheal is followed by a lingering papule which is attended by considerable itching. It is most commonly observed in children.

Urticaria Hemorrhagica.—The lesions are infiltrated with blood.

Urticaria Tuberosa (Giant Urticaria).—In this form the wheals may reach the size of an egg.

DIAGNOSIS. *Erythema Multiforme and Erythema Nodosum.*—In both of these affections the lesions last much longer, and are free from itching.

PROGNOSIS.—Favorable. In some cases it tends to become chronic.

TREATMENT.—The cause should be removed when possible. In gastric irritation bismuth, or calomel and soda are useful.

When there is constipation a saline laxative may prove very efficient. The special remedies usually recommended are alkalies, salicylate of sodium, quinine, iodide of potassium, and atropine.

Locally, lotions of water and alcohol, carbolic acid, boric acid, or hydrocyanic acid are very useful:

℞ Acid. carbolic., ʒj–ʒij;
Glycerini, fʒss;
Alcohol., fʒvj;
Aquæ, q. s. ad Oj.—M.

Urticaria Pigmentosa.

This is a form of urticaria observed in young children. It is characterized by an eruption of wheals which are itchy and persistent, and which leave behind a yellowish or brownish pigmentation. The disease runs a chronic course of months or years.

HERPES SIMPLEX.

(Fever Blisters.)

DEFINITION.—An acute non-contagious disease, characterized by groups of small vesicles mounted on inflammatory bases.

ETIOLOGY.—Herpes is very common in febrile diseases, especially pneumonia, influenza, malaria, and cerebro-spinal meningitis. Local irritation also predisposes to it. It is dependent upon a peripheral toxic neuritis.

SYMPTOMS.—One or more clusters of small vesicles appear, usually on the face or genitalia. The vesicles are mounted on an inflammatory base, contain clear fluid, and show no tendency to rupture. Soon their contents become puriform, dry up, and form reddish-brown crusts which fall off in a few days. Burning and tingling precede and accompany the eruption.

VARIETIES.—When it appears on the face, it is termed *herpes facialis;* on the genitals, *herpes progenitalis.*

DIAGNOSIS.—Herpes progenitalis must be distinguished from *chancroid.* The history, the superficial character of the lesion, the burning pain, and the subsequent course will indicate herpes.

TREATMENT.—The lesion may be painted with flexible collodion, or the following lotion employed:—

℞ Zinc. oxid., gr. xv;
Glycerini, ♏ xv;
Liq. plumbi subacetat. dil., ♏ x;
Liq. calcis, ʒvj–ʒj.—M. (TILBURY FOX.)

Sig.—Apply locally.

HERPES ZOSTER.

(Zona, Shingles.)

DEFINITION.—An acute inflammatory disease characterized by groups of small vesicles mounted on inflammatory bases, associated with neuralgic pain, and following the distribution of certain nerve-trunks.

ETIOLOGY.—The disease commonly depends upon a peripheral neuritis. Injury, exposure to cold, and damp clothes predispose to it.

SYMPTOMS.—Clusters of vesicles mounted on inflammatory bases may appear on any part of the body; but they are most frequently observed along the course of the intercostal nerves. Only one side is affected. Sharp neuralgic pain precedes and accompanies the eruption. The fluid in the vesicles soon be-

comes turbid, dries up, and forms yellowish-brown crusts which fall off in a few days.

PROGNOSIS.—Favorable.

TREATMENT.—Tonics are often indicated. Bulkley recommends phosphide of zinc in doses of one-third of a grain every three hours. Morphine is sometimes required for the relief of pain. Phenacetine, however, usually gives relief.

Locally.—Sedative applications are required; the best are flexible collodion with morphine, or a solution of menthol or carbolic acid, followed by a dusting-powder of oxide of zinc or starch.

℞ Morph. sulph., gr. viij;
Collodii, f℥j.—M.
Sig.—Apply with a camel's-hair brush.

HERPES IRIS.

DEFINITION.—An inflammatory disease, characterized by groups of vesicles arranged in concentric rings which present a somewhat variegated appearance.

ETIOLOGY.—The causes are unknown. The disease is rare.

SYMPTOMS.—One or more rings of vesicles successively appear around a central vesicle or papule. The different ages of the rings which compose the patch impart to the latter a variegated appearance. Burning and itching are often attendant symptoms. The hands, arms, and feet are the parts most frequently affected. The lesions appear in successive crops over a period of several weeks. In some instances the vesicles are quite large and resemble the blebs of pemiphigus.

PROGNOSIS.—Favorable, but recurrent attacks are common.

TREATMENT.—The same as in herpes zoster.

ACNE.

(Acne Vulgaris.)

DEFINITION.—An inflammatory disease of the sebaceous glands, characterized by papules and pustules and usually seated on the face or back.

Etiology.—It generally develops about puberty. Anæmia, menstrual disorders, and gastro-intestinal disturbances predispose. Certain drugs like iodide and bromide of potassium and copaiba may induce the disease.

Pathology.—Acne lesions result from the irritation excited by retained sebaceous matter, hence the papules and pustules are commonly associated with blackheads, or comedones.

Symptoms.—An aggregation of small papules, pustules, and comedones about the face, chest, and shoulders. Pustules or papules predominate according as the disease is acute or chronic. New lesions develop as the old disappear, so that the disease usually runs a protracted course. Subjective phenomena are absent.

Varieties. *Acne Papulosa.*—In this form the lesion reaches the papular stage and advances no further.

Acne Pustulosa.—In this variety the papules develop into pustules.

Acne Indurata.—The inflammation is deeply seated, the base of the papule or pustule is firm, and the lesion is sluggish.

Acne Atrophica.—In this form the lesions are followed by small scars or pits.

Acne Hypertrophica.—In this form there is an overgrowth of connective tissue and the skin becomes thickened.

Diagnosis.—The distribution, the chronic character of the affection, the involvement of the sebaceous glands, and the association with comedones are the diagnostic features which separate acne from all other affections.

Prognosis.—Curable under persistent treatment.

Treatment.—The general health must be improved. The diet should be nutritious, but easily assimilable; rich food must be prohibited. Constipation should be relieved by mild laxatives. In the anæmic and debilitated iron, quinine, strychnine, and cod-liver oil are useful remedies. The special drugs which have been recommended are arsenic, ergot, and calx sulphurata. Arsenic is best suited to the sluggish indurated forms; and calx sulphurata (gr. $\frac{1}{10}$–$\frac{1}{6}$ four times daily) to the pustular variety.

Local Treatment.—In the acute form mild applications should be employed, like the following calamine lotion :—

℞ Pulv. zinc. oxid., ʒiij;
Pulv. calaminæ, ʒij;
Glycerini, fʒij;
Aquæ calcis, fʒvj.—M.

In chronic cases the sebaceous plugs should be removed by a watch-key and the pustules incised. Thorough washing with very hot water and green soap is also advisable. The best local remedies are sulphur, mercury, and resorcin.

℞ Calcis, ℥ss;
Sulphur. sublimat., ℥j;
Aquæ, ℥x.—M. (Vleminckx.)

Evaporate to six ounces and filter.

Sig.—Apply at first well diluted and gradually increase the strength.

Or—

℞ Sulphur. præcip., ʒj;
Ung. aquæ rosæ,
Petrolat. moll., āā ʒiv.—M. (Van Harlingen.)

Sig.—Apply night and morning.

Or—

℞ Hydrarg. ammoniat., gr. xx–xl;
Ung. aquæ rosæ, ℥j.—M.

Sig.—Use night and morning.

Or—

℞ Hydrarg. chlor. corrosiv., gr. ss–ij;
Emuls. amygdal. amar., fʒiv;
Tinct. benzoin. comp., fʒj.—M.

Sig.—Use locally.

ACNE ROSACEA.

Definition.—A chronic affection, usually located on the face in the region of the nose, and characterized by marked hyperæmia, dilatation of the vessels, overgrowth of tissue, and acne lesions.

Etiology.—Anæmia, menstrual disorders, gastric disturbances, exposure to extremes of temperature, and intemperance are the usual predisposing causes.

Symptoms.—The affected area is of a deep-red color; the vessels are dilated; the skin is thickened and lumpy, and

acne lesions coexist. In advanced cases the nose may become extremely large and lobulated (Rhinophyma).

Subjective phenomena are generally absent.

Diagnosis. *Lupus Vulgaris.*—In this disease there are soft pale-red papules, ulceration, and cicatrization, and no enlargement of the bloodvessels.

Prognosis.—Unless the hypertrophy is marked, the disease is curable under protracted treatment.

Treatment.—The general treatment is the same as in acne vulgaris.

Local Treatment.—Sulphur and mercury are the most reliable remedies. Vleminckx's solution is very useful. Dilated vessels should be destroyed by electrolysis. Large hypertrophies may be removed by the knife.

FURUNCULUS.

(Boil.)

Definition.—An acute, circumscribed inflammation of a sebaceous gland or hair-follicle, usually terminating in suppuration.

Etiology.—Single boils are usually due to local irritation. Their appearance in crops (Furunculosis) is usually indicative of impaired health. The entrance of pus cocci into the skin is always essential to their production.

Diagnosis.—Furuncles must be distinguished from *carbuncles;* the latter are single, large, flattened at their summits, and have multiple openings.

Treatment.—In furunculosis the cause should be searched for and, if possible, removed. Tonics like iron, quinine, cod-liver oil, and hypophosphites are often very useful. Calx sulphurata ($\frac{1}{10}$—$\frac{1}{6}$ gr. thrice daily after meals) sometimes proves serviceable. A solution of boric acid or of corrosive sublimate may be applied locally. The following paste will often abort them :—

Ichthyol,
Ung. hydrarg.,
Ext. belladonnæ, āā ʒj.—M.

Sig.—Apply locally and make pressure with strips of adhesive plaster.

CARBUNCULUS

(Anthrax.)

DEFINITION.—A circumscribed inflammation of the skin and deeper tissues, characterized by a dark-red, painful node which breaks down and evacuates through several apertures.

ETIOLOGY.—Lowered vitality from any cause predisposes. They are especially common in diabetes. The exciting cause is a special microbe.

SYMPTOMS.—A dark-red, painful, flattened node appears surrounded by a dusky-red area of induration. In a week or ten days suppuration begins, and the contents are discharged through several orifices. There is generally marked constitutional disturbance. The most common seats are the nape of the neck, back, and buttocks.

PROGNOSIS.—Guardedly favorable. Death is not an infrequent termination in the old and debilitated.

TREATMENT.—Generally tonics like quinine, iron, and whiskey are indicated. Opium may be required to relieve pain.

Local Treatment.—In the early stage they may be aborted by a central injection of ten to twenty minims of a 5 or 10 per cent. solution of carbolic acid in glycerine. When not seen until abortion is too late, firm compression may be made by straps applied concentrically, leaving the central orifice free for the discharge of sloughs; an antiseptic dressing may be applied over the straps.

PSORIASIS.

DEFINITION.—A chronic inflammatory disease, characterized by red, scaly, sharply-circumscribed, elevated lesions.

ETIOLOGY.—Psoriasis usually develops in young adults. Heredity, the gouty diathesis, pregnancy, and lactation seem to predispose. It is as common in the robust as in the debilitated. It is non-contagious.

PATHOLOGY.—A localized hypertrophy of the rete mucosum associated with inflammation.

Symptoms.—Little red spots appear on the body, and gradually grow until they reach the size of a dollar. The lesions are of a dull pink or red color, sharply defined, somewhat elevated, surrounded by healthy skin, and covered with abundant dry, pearly, overlapping scales. These scales are readily detached, leaving behind a dry, slightly excoriated surface. The lesions may be uniformly distributed over the entire body, but usually the extensor surfaces are more affected; a symmetrical arrangement is often observed. Itching is slightly or entirely absent. After a variable time the centre of the patch disappears and leaves behind a spot of healthy skin which gradually increases until no trace of the lesion remains. The disease runs a protracted course of months or years, improving in the summer and growing worse in the winter.

Diagnosis. *Eczema.*—In this disease the patches are not sharply defined, but shade off gradually into the surrounding skin; there is marked itching; there is usually a decided discharge, and healing begins at the periphery instead of at the centre as in psoriasis.

Seborrhœa.—In this affection the lesions are usually confined to the scalp and face, while psoriasis is general; the scales are gray and greasy; the patches are not circumscribed, and lack the inflammatory character of psoriasis.

Papulo-squamous Syphiloderm.—The history, the associated symptoms of syphilis, the coppery color of the lesions, the scant scaling, the special tendency to involve the hands and soles will render the diagnosis apparent.

Prognosis.—The disease disappears under treatment, but relapse generally follows after a longer or shorter period.

Treatment.—The general health may require attention. In the gouty alkalies are of value; and in the anæmic iron and cod-liver oil are indicated. Arsenic is often of considerable value; it should be given in small doses cautiously increased. Iodide of potassium (gr. x–xx thrice daily) is sometimes recommended.

Local Treatment.—The scales should be removed by alkaline baths before local applications are made. The best local remedies are tar, chrysarobin, salicylic acid, resorcin, sulphur, and ammoniated mercury.

℞ Acid. chrysophanic., gr. x;
Adipis benzoat., ʒj.—M.
Sig.—Apply twice daily.

Or—

℞ Sulphur. sublimat.,
Ol. cadini, āā ʒiv;
Sapon. virid.,
Adipis, āā ʒj;
Cretæ præp., ʒijss.—M. (WILKINSON.)

ECZEMA.

(Tetter.)

DEFINITION.—A non-contagious inflammatory disease of the skin, characterized by multiform lesions—erythema, papules, vesicles, pustules, scales, and crusts—and associated with infiltration, itching, and more or less discharge.

ETIOLOGY.—It is most common in the young and in the aged. Digestive disturbances, debility, gout, and rheumatism predispose to its development. It may be due to external irritants like cold, heat, the rhus-plant, hard soaps, certain dyes, etc.

PATHOLOGY.—Congestion and infiltration of the various layers of the skin.

VARIETIES.—E. erythematosum, E. papulosum, E. vesiculosum, E. pustulosum, E. squamosum, and E. rubrum.

Eczema Erythematosum.—This form consists in irregular patches marked by swelling, redness, and slight scaling, and accompanied by itching and burning. The most common seat is the face.

Eczema Papulosum.—In this form there is a close aggregation of minute acuminated papules accompanied by severe itching. It is frequently associated with the vesicular variety. The most common seat is the extremities.

Eczema Vesiculosum.—This consists in an ill-defined red patch surmounted by minute vesicles, and accompanied by intense itching. The vesicles soon rupture and leave a raw, weeping surface which becomes more or less covered with crusts. In children, it is most common on the face; in adults, on the extremities.

Eczema Pustulosum (*Eczema Impetiginosum*).—This consists in an aggregation of small pustules which break and lead to the formation of thick yellowish crusts. Itching is not marked. It is frequently associated with the vesicular variety. It is most commonly observed on the face and scalp of poorly-nourished children.

Eczema Squamosum.—In this form there are irregular ill-defined red patches accompanied by considerable scaling. It differs from the erythematous form in the large amount of scaling. Its most common seat is the scalp.

When there is a marked tendency to fissuring, as in *chapping*, this form is termed *eczema fissum;* and when there is a tendency to the formation of warty excrescences, it is termed *eczema verrucosum.*

Eczema Rubrum (*Eczema Madidans*).—This is a secondary variety and is recognized by a raw, dark-red, moist surface, more or less covered with thick yellowish-red crusts. The itching may be severe. In children it is frequently noted on the face, and in old people on the extremities.

Diagnosis. *Scabies.*—The history of contagion; the location of the lesions—between the fingers, on the wrists, under the mammæ, in the axillæ; and the presence of burrows will indicate scabies.

Psoriasis.—The sharply-defined patches, the dry scaling, the absence of marked itching, the symmetrical distribution, and the predilection for extensor surfaces will indicate psoriasis.

Acne Rosacea.—The presence of acne papules and pustules and of dilated bloodvessels, and the absence of itching will distinguish acne rosacea from erythematous eczema.

Seborrhœa.—The greasy scales and the absence of itching and of all inflammatory symptoms will indicate seborrhœa.

Sycosis.—The limitation of the lesions to the hair-follicles of the face and the absence of itching will distinguish sycosis from eczema.

Prognosis.—Generally favorable under persistent and judicious treatment.

Treatment. *General Treatment.*—The health must be improved. Tonics are frequently indicated. In strumous

children cod-liver oil may be of extreme value. Disturbances of the gastro-intestinal tract are frequently present, and will require appropriate treatment. In the gouty and rheumatic the alkaline mineral waters, colchicum, and the salts of lithium are indicated. Constipation must always receive attention. Of the special internal remedies, arsenic is the most important; it is, however, only indicated in the chronic cases in which bright redness, itching, and weeping are absent.

External Treatment.—In acute cases with marked inflammatory symptoms, soothing applications should be employed. A saturated solution of boric acid may be dabbed on for five or ten minutes, and may be followed by zinc ointment spread on lint; when there is much itching carbolic acid is very useful:—

℞ Acid. carbolic., ʒj;
Glycerini, ʒij;
Aquæ, q. s. ad fʒviij.—M.

Sig.—Apply locally.

The following is also frequently used:—

℞ Zinc. oxid., ʒss;
Pulv. calaminæ præp., ℈iv;
Glycerini, fʒj;
Liq. calcis, fʒvij.—M.

Sig.—Shake and apply locally.

In chronic cases crusts and scales should be removed by soap and water or by:—

℞ Saponis virid., ʒij;
Alcoholis, ʒj.—M.

Sig.—Apply thoroughly and remove with warm water.

The best external applications are salicylic acid, tar, mercury, and resorcin:—

℞ Acid. salicylic., gr. v–x;
Petrolat. moll., ʒiv;
Amyli,
Zinci oxid., āā ʒij.—M.
(Stelwagon and Duhring.)

Sig.—Apply twice daily.

Or—

℞ Hydrarg. ammoniati, ʒss;
Liq. picis alkalin., ʒj;
Ung. aquæ rosæ, ʒj.—M.

Or—

℞ Ol. cadini, f℥ss;
Glycerini, f℥j;
Ung. diachyli, f℥iiss.—M. (Tilbury Fox.)

Sig.—Apply locally.

LICHEN RUBER AND LICHEN PLANUS.

Lichen Ruber.—This is an extremely rare disease, characterized by the eruption of small, red, glazed, acuminated papules which show no tendency to coalesce, and which are associated with itching and failure of general health. The disease runs a chronic course, and may prove fatal through exhaustion.

Lichen Planus.—This form is characterized by an eruption on the extremities of small, red, flat papules which tend to spread, and by coalescing form dull-red, irregular patches. The lesions have an angular outline, are slightly umbilicated, and at first have a smooth and shiny appearance, but later are slightly scaly. There is more or less itching, but no impairment of the general health. As the old lesions disappear new ones take their place.

Etiology.—These affections are most frequently observed in poorly-nourished, middle-aged males.

Treatment.—The general health must be improved by good food and such tonics as iron, strychnine, and cod-liver oil. Arsenic is of considerable value. Locally, ointments of tar or mercury are useful.

Lichen Scrofulosis.

This is a chronic affection occurring chiefly in children of a strumous diathesis, and characterized by small, pale-red, or salmon-colored scaly papules. They tend to form in groups, and are most frequently observed on the trunk. Itching is absent. The disease runs a chronic course.

Treatment.—Remedies like iron, quinine, and cod-liver oil are indicated. Hebra recommends the last remedy as a local application.

PRURIGO.

DEFINITION.—A chronic inflammatory disease, characterized by a general eruption of minute, discrete papules, accompanied by marked itching.

ETIOLOGY.—It is most commonly observed in the poor and ill-fed of Europe. It develops in early childhood and persists through life.

SYMPTOMS.—An eruption of small, discrete, deeply-situated, pale-red papules appears on the body, especially on the back and extensor surfaces of the extremities. The skin is harsh, dry, and thickened, and covered with numerous scratch-marks induced by the intense itching.

PROGNOSIS.—Unfavorable; it usually persists through life.

TREATMENT.—The general health must be improved by good food and the use of nutrient tonics like iron and cod-liver oil. Frequent bathing, followed by ointments of tar, sulphur, or naphthol, gives relief.

DERMATITIS HERPETIFORMIS.

(Herpes Gestationis, Duhring's Disease.)

DEFINITION.—A chronic inflammatory disease, characterized by multiform lesions which form in groups, and which are associated with intense itching.

ETIOLOGY.—Women are more commonly affected than men. Pregnancy, lactation, and menstrual disorders seem to exert a predisposing influence.

SYMPTOMS. *Erythematous Form.*—This is characterized by the appearance in crops of erythematous patches which are associated with considerable itching.

Papular Form.—Groups of papules appear in crops, and are frequently associated with erythema, vesicles and scratch-marks.

Vesicular Form.—Groups of irregular-shaped vesicles resembling herpes appear in crops and are often associated with erythema, pustules, and scratch-marks.

Pustular Form.—This resembles the former, but the vesicles are replaced by pustules.

Bullous Form.—Large irregular-shaped blebs appear in crops and tend to group. Vesicles and patches of erythema are also frequently present.

Mixed Form.—Vesicles, erythematous patches, pustules, papules, and blebs appear in association, come out in crops, and are attended with intense itching.

In the pustular, bullous, and mixed forms there may be marked constitutional disturbances.

PROGNOSIS.—Guardedly favorable. The disease runs a chronic course. Relapses are very common.

TREATMENT.—Tonics are generally indicated. Lotions of boric or carbolic acid may be employed to allay itching, and may be followed by a dusting-powder.

DERMATITIS.

DEFINITION.—Inflammation of the skin resulting from the action of some irritant.

Dermatitis Traumatica.—This term is applied to inflammation of the skin resulting from traumatism.

TREATMENT.—The removal of the cause and the application of soothing remedies will usually suffice.

Dermatitis Venenata.—The term is applied to inflammation of the skin resulting from the application of vegetable, animal, or chemical irritants. Notable examples of this form of dermatitis are observed in susceptible people after exposure to the influence of poison ivy (*Rhus Toxicodendron*), poison oak (*Rhus Venenata*), or poison sumach (*Rhus Diversiloba*).

SYMPTOMS OF RHUS-POISONING.—The affection resembles acute eczema, and may appear in a few hours or not until the lapse of several days after exposure to the plant. It is generally observed on the face or hands. The part becomes red and swollen, and soon minute papules and vesicles appear. It gives rise to considerable burning and itching. As a rule, it subsides in a few days, but in patients with sensitive skin it may linger for several weeks.

TREATMENT.—The part should first be bathed with castile soap and tepid water, and then treated with some sedative lotion or ointment. Black wash may be dabbed on, and zinc

ointment subsequently applied; or a saturated solution of boric acid may be followed by zinc ointment. When there is marked itching a weak solution of carbolic acid (3j to Oj) is useful. The fluid extract of grindelia robusta has been highly recommended; it may be applied in the strength of half an ounce to a pint of water.

Dermatitis Calorica.—This term is applied to the inflammation of the skin resulting from extreme heat or cold. *Pernio*, or *chilblain*, is characterized by redness, swelling, intense burning and itching, and results from a sudden change from a low temperature to a high temperature. *Frost-bite* is characterized by congelation; the part is of a dull-white color and is anæsthetic; subsequently inflammation or gangrene develops.

Burns and *scalds* result from the application of heat, and are divided into degrees according to the depth to which the destructive process extends.

TREATMENT.—In *pernio*, or *chilblain*, the part should first be rubbed with snow or bathed in ice-water until the circulation is re-established; and then an application made of nitrate of silver (gr. v to the ounce of distilled water) or of tincture of iodine.

In superficial *burns* or *scalds* one of the following remedies may be applied: Phénol sodique, carron oil (equal parts of linseed oil and lime-water), powdered bicarbonate of sodium, or:—

℞ Acidi carbolic., gr. viij;
Vaselin., ʒij.—M. (BELLVUE HOSPITAL.)
Sig.—Spread on lint and apply where the skin is broken.

Dermatitis Medicamentosa.—This term is applied to the various cutaneous eruptions which follow the internal use of certain drugs.

Belladonna or Atropia.—These drugs produce a diffuse erythematous rash resembling that of scarlet fever, but it lacks the punctiform character of the latter. It usually appears on the face, neck, and chest, and is associated with dryness of the throat, rapid pulse, and if the dose has been large, dilated pupils.

Cubebs.—This drug sometimes produces an erythema associated with minute papules.

Copaiba.—The rash may be macular, papular, or like that of urticaria.

Bromide of Potassium.—The eruption resembles acne and consists of papules and pustules.

Iodide of Potassium.—The eruption may be erythematous, papular, pustular, urticarial, or purpuric. The most common eruption resembles acne, but the lesions are bright-red in color and widely distributed over the surface of the body.

Arsenic.—The eruption may be erythematous, papular, vesicular, or pustular.

Antipyrin.—This drug not infrequently produces a widespread papular eruption.

Quinine.—The rash is usually erythematous, though an urticarial eruption has been observed.

Salicyl Compounds.—The eruption is usually erythematous or urticarial.

Borax.—This drug occasionally produces an eruption resembling psoriasis.

Chloral.—The eruption is usually erythematous or urticarial.

Dermatitis Exfoliativa.

This is a rare affection, characterized by diffuse redness of the skin, high fever and its associated phenomena, and desquamation. It is interesting from its close resemblance to *scarlet fever*, from which it may be distinguished by the history and the absence of sore throat, and a "strawberry" tongue.

ECTHYMA.

Definition.—An inflammatory affection, characterized by the appearance of discrete, flat pustules, which vary in size from a pea to a five-cent piece, and which are surrounded by a distinct red areola.

Etiology.—Male sex, middle life, bad hygiene, and debility are predisposing factors.

Symptoms.—Flat, yellow pustules appear in crops. They are surrounded by a distinct red areola and soon dry up, forming reddish-brown crusts. Slight excoriation and pigmenta-

tion sometimes remain after the separation of the crusts. Subjective phenomena are usually absent.

Diagnosis.—The acute course, the absence of ulceration, and the absence of history and of associated symptoms of syphilis will separate it from the *pustular syphilide.*

Impetigo.—In this affection the lesions are not flat; they are not distinctly inflammatory; and the crusts are light yellow, not reddish-brown. Impetigo occurs most frequently in children, who may be quite robust.

Prognosis.—Favorable.

Treatment.—Constitutional treatment is generally required. Such tonics as iron, quinine, strychnine, and cod-liver oil are often idicated.

Local Treatment.—The crusts should be removed and some stimulating ointment applied, as the following:—

℞ Hydrarg. ammoniat., gr. x;
Ung. zinci oxidi, ℥j.—M.

PEMPHIGUS.

Definition.—A non-contagious inflammatory disease, characterized by the eruption of successive crops of bullæ or blebs.

Etiology.—Female sex, nervous prostration, heredity, and injury to the peripheral nerves are predisposing factors.

Varieties.—Pemphigus vulgaris and pemphigus foliaceus.

Pemphigus Vulgaris.—This form usually runs a chronic course and is characterized by successive crops of blebs, varying in size from a small pea to a large walnut. They are thoroughly distended with fluid, which is at first clear but subsequently turbid. As a rule, they do not rupture, but disappear in the course of five or six days, their contents being gradually absorbed. After absorption a thin pellicle remains, which dries and is subsequently detached, leaving behind a slightly pigmented spot. No part of the body is exempt; and as one set of blebs disappears, new ones rapidly develop, and so the disease continues for many years.

In severe cases there may be considerable itching and burning attending the eruption.

Pemphigus Foliaceus.—This rare and grave form of pemphigus is characterized by crops of blebs, which are flaccid and filled with a turbid fluid almost from the beginning. They soon rupture and form thick crusts, which separating leave behind red weeping surfaces. The crops follow each other in rapid succession, and at times the whole body may be covered with blebs and scabs. The disease may last several years, death ultimately resulting from exhaustion.

Diagnosis. *Bullous Syphiloderm.*—The history, the associated symptoms of syphilis, the thick, yellow, stratified crusts, and the underlying ulceration will serve to separate this affection from pemphigus.

Impetigo Contagiosa.—The acute course, the contagious and auto-inoculable character of the affection, and the umbilication of the blebs will separate impetigo contagiosa from pemphigus.

Prognosis.—The prognosis should be guarded. Pemphigus vulgaris runs a long course and is often intractable. Pemphigus foliaceus often proves fatal through exhaustion.

Treatment.—The diet should be nutritious, but carefully adapted to the stomach. The patient should be placed under the best hygienic conditions. Tonics like iron, quinine, phosphorus, cod-liver oil, and strychnine are usually indicated. In many cases arsenic is a valuable remedy.

Local Treatment.—The blebs may be punctured and subsequently dressed with zinc ointment.

IMPETIGO.

Definition.—An acute inflammatory disease, characterized by an eruption of discrete pustules varying in size from a pea to a cherry.

Etiology.—The exciting cause is unknown. It is most commonly observed in children.

Symptoms.—A pustular eruption appears generally on the face and extremities. The pustules are generally few in number, and are discrete, tense, and surrounded by a slight areola. In a few days they dry up and form thin yellowish-brown

crusts, which soon drop off and leave behind a normal surface. Subjective phenomena are absent.

DIAGNOSIS. *Ecthyma.*—This affection occurs most frequently in debilitated adults; the pustules are flat, surrounded by a distinct areola, and dry to brown crusts which separate and leave a pigmented excoriated surface.

Impetigo Contagiosa.—As the name implies, this affection is contagious and is auto-inoculable; its pustules are flat and umbilicated, and dry up and form lamellated, thin, yellow crusts.

PROGNOSIS.—Favorable. It terminates spontaneously in a few days or a week.

TREATMENT.—Open the pustules and apply some simple protective ointment, like that of oxide of zinc.

IMPETIGO CONTAGIOSA.

DEFINITION.—An acute contagious inflammatory disease, characterized by flat, yellowish blebs which dry up and form thin, yellow, lamellated crusts.

ETIOLOGY.—Its exciting cause is unknown. It is most frequently observed in debilitated children.

SYMPTOMS.—The eruption is most frequently observed on the face and extremities; it generally appears in crops, and is at first vesicular. The vesicles grow, and are soon converted into flat, umbilicated pustules which vary in size from a pea to a large walnut. They have a slight red areola. Itching is slight or entirely absent. In some cases there is moderate fever with its associated phenomena. In a few days the blebs dry up and form thin, yellow, lamellated crusts which separating leave a slightly excoriated surface. The disease is contagious, and the lesions are auto-inoculable.

DIAGNOSIS. *Eczema.*—In this disease the pustules are deeper, more confluent, excite intense itching, and are associated with inflammation and infiltration of the surrounding skin.

Simple Impetigo.—This affection is not contagious or auto-inoculable; the pustules are tense, not flat or umbilicated; and the subsequent crusts are yellowish-brown and are not followed by excoriation.

PROGNOSIS.—Favorable. It terminates spontaneously in a few days or weeks.

TREATMENT.—A slight stimulating ointment like the following is sometimes useful :—

℞ Hydrarg. ammon., gr. x–xx;
Adipis, ʒj.—M.
Sig.—Apply to the surface after removal of the crusts.

MILIARIA.

(Prickly Heat.)

DEFINITION.—An acute inflammatory disease of the sweat-glands, characterized by a discrete eruption of minute papules and vesicles.

ETIOLOGY.—Childhood and high temperature are the principal predisposing causes.

SYMPTOMS.—The eruption generally appears on the trunk, and consists of minute closely-aggregated red papules or clear vesicles. The lesions are discrete, and excite some burning and itching. It is generally associated with free perspiration.

DIAGNOSIS.—*Eczema papulosum* differs from miliaria in that the papules are larger, appear more gradually, disappear more slowly, and excite intense itching.

Eczema vesiculosum differs from miliaria in that the vesicles are large, disappear more slowly, show a tendency to break, and are associated with marked itching.

Sudamen differs from miliaria in that it lacks all inflammatory characteristics.

PROGNOSIS.—Favorable. Obstinate cases may persist for several weeks.

TREATMENT.—The general health may require attention. The diet should be light, and easily assimilable. Constipation should be relieved by saline laxatives. Locally, a simple dusting-powder is generally all that is required.

℞ Pulv. amyli, ʒvj;
Zinc. oxidi, ʒiss;
Pulv. camph., ʒss.—M. (HARDAWAY.)
Sig.—Dusting-powder.

Or—

℞ Zinc. carbonat. præcip., ʒiv;
Zinc. oxidi, ʒij;
Glycerini, fʒij;
Aq. rosæ, fʒviij.—M. (Tilbury Fox.)

Sig.—Apply locally.

ALBINISM.

Definition.—A congenital deficiency of pigment.

Etiology.—Beyond heredity, no cause is known. Partial albinism is more common in the negro.

Symptoms.—In complete albinism the skin is white; the hair is thin, soft, and very light in color; the pupils appear red, the eyes are very sensitive to light, and the iris and choroid are deficient in pigment.

VITILIGO.

(Leucoderma.)

Definition.—An acquired cutaneous affection, characterized by milk-white patches which are surrounded by areas of increased pigmentation.

Etiology.—The disease seems to be more common in the tropics and in the colored race. The condition probably results from disturbed innervation.

Symptoms.—Milk-white spots appear on the body and grow very slowly; their borders usually reveal an increase of the normal pigment. Apart from the absence of pigment the skin is normal.

Diagnosis. *Morphœa.*—The initial hyperæmia and the subsequent atrophy of the skin will serve to distinguish morphœa from vitiligo.

Anæsthetic Leprosy.—The subjective symptoms, the atrophy of the tissues, and the anæsthesia will separate leprosy from vitiligo.

Prognosis.—Unfavorable; the disease usually persists through life.

Treatment.—Tonics and local stimulants may be tried. Among the latter, electricity, blisters, and irritating ointments have been recommended, arsenic is recommended.

CANITIES.

Definition.—Grayness of the hair.

Etiology.—Local grayness may be congenital, or result from some disturbance of innervation, as in neuralgia of the supraorbital nerve. As a general condition it is usually an expression of senility, although it occasionally develops very early in life. Profound emotional disturbances sometimes induce an abrupt development of canities.

Prognosis.—The condition is permanent, and treatment is of no avail.

ATROPHY OF THE SKIN.

Etiology.—Atrophy of the skin occurs under several conditions. A local atrophy may result from inflammation or injury of a nerve-trunk; in these cases, the wrinkles are absent, the skin is thin, smooth, and shiny, and there is often intense burning in the part ("*glossy skin*"). Atrophy is also observed in leprosy, morphœa, and scleroderma.

Universal atrophy of the skin results from senility, and very rarely as an idiopathic condition. Sometimes the atrophy occurs in lines or spots (*striæ et maculæ atrophicæ*) as an idiopathic condition, or as the result of stretching the skin, as in the *lineæ albicantes* following pregnancy.

ATROPHY OF THE HAIR.

Etiology.—Atrophy of the hair may result from local diseases which interfere with the nutrition of the scalp, such as seborrhœa, eczema, ringworm, etc.; or it very rarely arises as an idiopathic condition without obvious cause.

Prognosis.—When the cause can be ascertained and removed, the prognosis is favorable.

Treatment.—Local diseases will require appropriate treatment. The general health should be improved. Stimulating applications of mercury, sulphur, or carbolic acid are sometimes useful.

ATROPHY OF THE NAILS.

Etiology.—Occasionally the condition is congenital, but more frequently it is acquired, and results from injury or disease of the nerve-trunk; from some general disease, like one of the fevers, syphilis, or cancer; or from some disease of the skin, as psoriasis or ringworm.

Symptoms.—The nails lose their lustre, cease to grow, and become opaque and brittle.

Prognosis and Treatment.—Both will depend on the exciting cause.

ALOPECIA.

(Baldness.)

Etiology.—(1) Baldness may be congenital; in these cases it is usually partial. (2) It may be an expression of senility; in which case it generally begins on the crown or brow, and is associated with more or less atrophy of the scalp. (3) It may occur early in life, as an idiopathic affection arising without obvious cause. (4) It may result from general diseases, like syphilis and the fevers. (5) In early life it is often due to some local disease, especially seborrhœa.

Prognosis.—In congenital, senile, and idiopathic alopecia the prognosis is unfavorable. In the alopecia of general diseases the prognosis is usually favorable. In alopecia resulting from seborrhœa much can be accomplished by persistent and judicious treatment.

Treatment.—The general health should be improved. Frequent washing the head with warm water and castile soap is to be recommended. One of the following local stimulants may be prescribed: Cantharides, quinine, alcohol, capsicum, sulphur, or carbolic acid.

℞ Quininæ sulph., ʒss;
Tinct. cantharidis, fʒj;
Spt. ammon. aromat., fʒj;
Ol. ricini, fʒiss;
Spt. myrciæ, fʒvss;
Ol. rosmarini, gtt.v.—M. (Gerhard.)

Or—

℞ Tinct. cantharidis, f℥j ;
Acid. carbolici, ʒj ;
Ol. ricini, ʒiss ;
Spt. myrciæ,
Spt. lavandulæ, āā f℥ij.—M.

Or—

℞ Tinct. cantharidis, f℥ij ;
Quininæ sulph., gr. x ;
Glycerini, f℥ss ;
Ol. rosmarini, gtt. v ;
Spt. myrciæ, q. s. ad f℥v.—M.

ALOPECIA AREATA.

(Alopecia Circumscripta.)

Definition.—Baldness appearing in circumscribed patches without any obvious lesion of the skin.

Etiology.—The cause is unknown. Some regard it as of parasitic origin, while others look upon it as a neurosis. It is generally observed in early adult life.

Symptoms.—The disease is characterized by the sudden or gradual appearance of circumscribed round patches of baldness. At first there is no change in the appearance of the skin, but later it may become pale and atrophied. Although the scalp is the most frequent seat, it occasionally involves other hairy parts, as the eyebrows, beard, etc.

Diagnosis. *Ringworm.*—Ringworm is exceedingly rare in adults, and is characterized by elevated scaly patches through which project dry, brittle, broken hairs. If there should be any doubt in the diagnosis, the microscope may be employed to detect the tricophyton.

Prognosis.—In the majority of cases the hair returns under prolonged and persistent treatment. The older the patient the less favorable the prognosis.

Treatment.—General tonics like iron, arsenic, quinine, and strychnine are usually indicated. The local treatment should be stimulating and consist in the application of blisters, elec-

tricity, friction, rubefacient liniments, or ointments containing chrysarobin, tar, sulphur, or ammoniated mercury.

℞ Tinct. cantharidis,
Tinct. capsici, āā f℥iss ;
Olei ricini, f℥ij ;
Alcoholis, f℥vj ;
Spts. rosmarini, f℥ij.—M.
(DUHRING and STELWAGON.)

Or—

℞ Acid. chrysophanic., ʒiss ;
Adipis, ℥ij.—M.

Or—

℞ Sulphur. loti, ʒiv ;
Ol. cadini, ʒij ;
Adipis, ℥j.—M.

SYCOSIS.

(Simple Sycosis, Folliculitis Barbæ.)

DEFINITION.—A non-contagious inflammatory disease of the hair-follicles.

ETIOLOGY.—The affection results from local irritation and the entrance of pyogenic cocci.

SYMPTOMS.—The disease usually manifests itself on the bearded region of the face, and is characterized by an aggregation of papules and pustules, each of which is pierced by a hair. When the lesions are discrete the intervening skin is often quite healthy ; but when they are close together it is often infiltrated and hyperæmic. During the papular stage the hairs are not loose, but firmly attached ; during the pustular stage, however, they can be readily extracted. The pustules show no tendency to rupture, but dry to yellowish-brown crusts. Acute cases are associated with more or less burning and itching. If the disease persists, it may lead to extreme destruction of the hair-follicles and, as a consequence, to permanent alopecia.

DIAGNOSIS. *Eczema.*—The lesions in eczema excite severe itching, are not perforated by hairs, and are not confined to the hairy parts.

Tinea Sycosis, or Barber's Itch.—The affection begins as a

red scaly patch, and is followed by the development of large, deeply-seated tubercles. The hairs soon become dry, brittle, and broken off, and can be easily extracted. In doubtful cases the microscope may be employed for the detection of the tricophyton.

PROGNOSIS.—The disease is curable under prolonged and judicious treatment. Relapses are very prone to occur.

TREATMENT.—In acute cases soothing applications are indicated; thus the parts may be dabbed with black wash or a saturated solution of boric acid, and subsequently spread with oxide of zinc ointment. In chronic cases the crusts should be removed, and the hairs cut close or preferably, shaved. It is advisable to puncture the pustules and to extract the hairs, so as to preserve the follicles. When the parts are not irritable stimulating applications are useful, and one of the following may be selected:—

℞ Sulphur. præcip., ʒij;
Ung. aquæ rosæ, ℥j.—M.
Sig.—Apply twice daily.

Or—

℞ Ung. diachyli,
Ung. zinc. oxidi, aa ℥iss;
Ung. hydrarg. ammon., ʒiij;
Bismuth. subnitratis, ʒiss.—M. (ROBINSON.)
Sig.—Apply twice daily.

Or—

℞ Ichthyol., ʒj;
Ung. diachyli, ℥j;
Sig.—Apply twice daily.

POMPHOLYX.

(Dysidrosis.)

Pompholyx is a very rare disease, usually observed in those who perspire freely, and characterized by an eruption of deeply-seated vesicles which resemble sago-grains imbedded in the skin. The vesicles most commonly appear on the hands, especially between the fingers, and gradually increase in size

until they reach the dimensions of blebs. They show no tendency to rupture, but dry up, and are followed by extensive desquamation of the cuticle. The eruption often excites considerable pain and tenderness. The disease usually disappears in the course of a few weeks, but is prone to recur.

TREATMENT.—General tonics like iron, strychnine, and arsenic are often indicated. Locally, sedative lotions or ointments should be employed.

LENTIGO.

(Freckle.)

DEFINITION.—A deposition of pigment in the form of small, irregular-shaped brownish spots.

ETIOLOGY.—Blondes are more subject to the affection than brunettes. Exposure to the sun's rays often serves as an exciting cause.

SYMPTOMS.—Exposed parts—the face, shoulders, arms, and hands—are mostly affected. The patches vary in color from yellow to dark brown, and range in size from a pin-head to a pea.

PROGNOSIS.—Freckles can be removed, but they always return.

TREATMENT.—One of the best remedies is the bichloride of mercury in solution or ointment.

℞ Hydrarg. chlor. corros., gr. iv;
Alcohol. et aquæ, āā ad f℥iv.—M.
Sig.—Apply twice daily.

CHLOASMA.

DEFINITION.—An abnormal deposition of pigment in the form of large brown or liver-colored patches.

ETIOLOGY.—It may result from the application of external irritants; from general diseases like malaria and Addison's disease; or from affections of the uterus, as pregnancy, tumors, etc.

SYMPTOMS.—The affection consists in the appearance—especially on the face—of large, round, or irregular-shaped

brownish or blackish patches. Apart from the discoloration the skin is normal.

Diagnosis.—In *Leucoderma* the periphery of the patches is pigmented, but the central milk-white appearance is not seen in chloasma.

Prognosis.—When the cause can be removed the prognosis is favorable.

Treatment.—When possible the cause should be removed. The best local remedies are bichloride of mercury and sulphur.

℞ Zinci oxidi, gr. iij ;
Hydrarg. ammoniat., gr. iss ;
Ol. theobrom.,
Ol. ricini, āā ʒiiss ;
Essent. rosæ, gtt. x.—M. (Monin.)

Sig.—Apply to the face night and morning.

KERATOSIS PILARIS.

(Lichen Pilaris.)

Definition.—Small, papular elevations resulting from hypertrophy of the epidermis surrounding the outlets of the hair-follicles.

Etiology.—It generally results from infrequent bathing.

Symptoms.—The skin, particularly on the extensor surfaces of the arms and legs, is the seat of numerous pin-head elevations, which have a dirty-gray color and are pierced by hairs. It may excite some itching. Generally there are no evidences of inflammation, but sometimes a few red papules or even pustules result from irritation.

Diagnosis.—In *Cutis Anserina*, or goose-flesh, the lesions are transient and have the color of normal skin.

Prognosis.—Favorable.

Treatment.—In most cases nothing will be required beyond frequent bathing with soap, followed by friction of the skin. In obstinate cases some simple ointment may be applied after bathing.

MOLLUSCUM EPITHELIALE.

(Molluscum Contagiosum, Molluscum Sebaceum.)

DEFINITION.—A cutaneous affection, characterized by the appearance of discrete wax-like elevations ranging in size from a pin-head to a pea, and varying in color from white to rose-pink.

ETIOLOGY.—The disease is generally observed in children, and frequently affects several members of the same household, school, or asylum. It is probably contagious.

SYMPTOMS.—Small white or pale-pink, wax-like elevations appear, especially on the face. They are always discrete and rarely abundant. The centre of the elevation is depressed and reveals a dark spot which corresponds to the aperture of the follicle. At first the lesions are quite firm, but as they grow old they become soft. When firmly squeezed they exude a soft, cheesy material. After remaining for several weeks they break down or undergo slow absorption.

DIAGNOSIS.—The color, the wax-like appearance, the umbilication, and the central aperture are the diagnostic features.

PROGNOSIS.—Favorable, although the disease may run a protracted course of months or years.

TREATMENT.—General tonics like iron, strychnine, and arsenic are often indicated. The lesions should be incised, the contents expressed, and their bases touched with nitrate of silver; ointments of mercury and sulphur have also been recommended.

CALLOSITAS.

(Callus, Keratoma, Tylosis.)

DEFINITION.—A thickened, horny condition of the skin resulting from hypertrophy of the corneous layer of the epidermis.

ETIOLOGY.—Constant irritation from friction or pressure is the chief cause; hence it is frequently seen on the feet from the rubbing of shoes, and on the hands from the friction of tools.

Symptoms.—The condition is characterized by the appearance of hard, thickened, grayish masses, which gradually merge into healthy skin. The soles and palms are the parts most frequently affected. When slight it causes little inconvenience, but occasionally it becomes fissured and painful.

Prognosis.—It yields rapidly to treatment when the cause is removed.

Treatment.—When excessive the parts should be soaked and the thickened epidermis pared off. One of the best remedies for softening the horny overgrowth is salicylic acid; it may be applied in the form of a plaster or in collodion.

℞ Acid. salicylic., ʒj;
Collodii, fʒj.—M.
Sig.—Apply night and morning.

CLAVUS.

(Corn.)

Definition.—Clavus is a circumscribed thickening of the epidermis usually appearing on the feet.

Etiology.—Corns generally result from the friction of ill-fitting shoes.

Symptoms.—Small, circumscribed, horny elevations appear upon the feet and often excite severe pain. When bathed in perspiration they become more or less macerated, and in this condition constitute the so-called *soft corn*.

Treatment.—A radical cure requires the use of well-fitting shoes. The corns may be removed by soaking, paring, and the application of some mild caustic like salicylic acid.

℞ Acid. salicylic., gr. xxx;
Tinct. iodi, ♏x;
Ext. cannabis ind., gr. x;
Collodii, fʒss.—M.
Sig.—Apply night and morning for several days, and then soak in hot water.

CORNU CUTANEUM.

(Cutaneous Horn.)

DEFINITION.—A circumscribed, projecting outgrowth resulting from hypertrophy of the epidermis.

SYMPTOMS.—Horns generally appear on the face, scalp, or penis, and are usually observed in the old. They consist of dry, rough, horny, more or less conical projections, which vary in length from a few lines to several inches.

PROGNOSIS.—Favorable.

TREATMENT.—The horn should be excised and the base subsequently cauterized.

VERRUCA.

(Wart.)

DEFINITION.—A wart is a circumscribed elevation resulting from hypertrophy of the papillæ and epidermis.

ETIOLOGY.—The cause is obscure. A bacterial origin has been suggested. They are most frequently observed in children.

SYMPTOMS.—*Verruca Vulgaris*, or common wart, is generally observed on the hands of children. It consists of a firm, circumscribed elevation, varying in size from a millet-seed to a pea.

Verruca plana, or flat wart, is a circumscribed, flat, pigmented elevation usually observed on the backs of old people.

Verruca Filiformis.—This is a thread-like overgrowth, and is generally observed on the soft parts, like the face and neck.

Verruca Digitata.—This form is made up of numerous branches, and is generally observed on the scalp.

Verucca Acuminata, or Venereal Wart.—This appears in groups about the genitalia. It is soft, red in color, and highly vascular. It may be dry or moist according to its location; the latter condition often gives rise to a peculiarly offensive odor.

TREATMENT.—Ordinary warts may be removed by excision, caustics, or electrolysis.

Venereal warts should be bathed in some antiseptic solution and subsequently dusted with calomel, iodoform, or boric acid.

NÆVUS PIGMENTOSUS.

(Mole.)

DEFINITION.—A circumscribed deposit of pigment, usually associated with hypertrophy of cutaneous structures.

ETIOLOGY.—Moles are usually congenital.

SYMPTOMS.—The neck, face, and trunk are favorite localities. The nævi vary in number from one to several hundred; in size, from a millet-seed to a filbert; and in color, from yellow to black. When the surface is smooth, the growth is termed *nævus spilus;* when the surface is covered with hair, it is termed *nævus pilosus;* when the surface is warty, it is termed *nævus verrucosus;* and when there is much overgrowth of connective tissue, it is termed *nævus lipomatodes.*

TREATMENT.—They may be removed by excision, the aplication of caustics, or by electrolysis.

ICHTHYOSIS.

(Fish-skin Disease.)

DEFINITION.—A chronic affection characterized by dryness, thickening of the epidermis, and scaliness.

ETIOLOGY.—The affection is often hereditary and is usually detected in early childhood.

SYMPTOMS.—The skin is dry and harsh; the surface is covered with adherent polygonal scales; and the papillæ are more or less hypertrophied. The term *Ichthyosis hystrix* is applied to the condition when there is excessive hypertrophy of the papillæ. The extensor surfaces of the extremities are the parts most involved.

DIAGNOSIS.—The absence of all inflammatory symptoms will separate ichthyosis from *squamous eczema* and *psoriasis.*

PROGNOSIS.—The disease is incurable; but the patient can be rendered comfortable by appropriate treatment.

TREATMENT.—The scales may be removed by alkaline baths or by applications of green soap. The skin may be rendered pliable by rubbing in some simple ointment.

℞ Sulphuris, gr. xxv–l;
Ung. simp., ℥j.—M. (UNNA.)
Sig.—Rub in at night.

ONYCHAUXIS.

Onychauxis, or hypertrophy of the nail, may be congenital, or may result from certain skin affections, such as eczema, ringworm, or syphilis; from diseases of the nerves, as neuritis; or from traumatism.

HYPERTRICHOSIS.

(Hirsuties.)

Hypertrichosis, or hypertrophy of the hair, may be local or general. The term is applied not only to an excessive overgrowth of hair, but to a growth of hair in unusual localities, as on the faces of young women.

TREATMENT.—The hair may be removed temporarily by shaving, epilation, or depilatories. Permanent relief can only be accomplished by electrolysis.

SCLERODERMA.

(Sclerema, Scleriasis.)

DEFINITION.—A pigmented, rigid, indurated condition of the skin, occurring in circumscribed patches or involving the entire body.

ETIOLOGY.—The cause is unknown.

SYMPTOMS.—The affection may be diffuse or involve circumscribed patches. It may appear quite suddenly, or develop very gradually in the course of months or years. The skin assumes a yellowish-brown color, becomes rigid, indurated, and hide-bound; the surface is unnaturally dry and smooth. When the condition is advanced the joints become more or less immobile.

Prognosis. — Guarded. It often recovers spontaneously after having persisted for a long time. In other cases the process may spread until the patient becomes almost helpless.

Treatment.—Tonics like iron, arsenic, and cod-liver oil are often indicated. Locally, massage, friction, electricity, and inunctions are recommended.

MORPHŒA.

(Addison's Keloid.)

Definition.—A cutaneous affection, characterized by circumscribed, rounded, ivory-like patches, which have hyperæmic or pigmented borders.

Etiology.—The cause is unknown. By many it is regarded as a circumscribed form of scleroderma.

Symptoms.—The lesions usually appear upon the trunk and consist of sharply-circumscribed patches, which are at first slightly hyperæmic. The surface is smooth and resistant to the touch. As the patch grows old its centre becomes pale and ivory-like, while the periphery remains hyperæmic or becomes pigmented.

Prognosis.—Guarded.

Treatment.—The same as scleroderma.

ELEPHANTIASIS.

(Elephantiasis Arabum, Elephantiasis Pachydermia, Barbadoes Leg.)

Definition.—Hypertrophy of the skin and subcutaneous tissues, usually associated with lymphangitis, œdema, and pigmentation.

Etiology.—While elephantiasis may occur in any part of the world, it is far more common in the tropics. It is most frequently observed in the male sex, and rarely develops before adult life. It results from obstruction of the lymphatics, and the most common cause of such obstruction is the presence of a parasite—*filaria sanguinis hominis.*

PATHOLOGY.—Examination of the affected tissues reveals hypertrophy of the connective tissue, œdema, and inflammation and dilatation of the lymphatic vessels.

SYMPTOMS.—It usually begins with recurring attacks of erysipelatoid inflammation. The part is red, swollen, and painful; the lymphatics may be traced as branching red lines beneath the skin; and with these local phenomena there is more or less fever. After each attack the part is left a little enlarged, until finally it presents the following characteristic appearance: it is enormously swollen; the skin is thickened, roughened, and pigmented; and the papillæ are unusually prominent. The regions generally affected are the legs and genitals. In elephantiasis of the scrotum (*lymph-scrotum*) the hypertrophied mass may weigh as much as fifty or even a hundred pounds.

PROGNOSIS.—In the early stage the disease may be arrested, but when fully established it is incurable.

TREATMENT.—The acute inflammatory attacks should be treated by rest and the application of sedative lotions, like lead-water and laudanum. Subsequently mercurial inunctions may be employed, and the part firmly bandaged with the view of promoting absorption. Amputation may be successfully employed in lymph-scrotum. In elephantiasis of the limbs ligation of the main artery has given somewhat encouraging success. More recently galvanism has given very good results.

DERMATOLYSIS.

(Pachydermatocele, Cutis Pendula.)

DEFINITION.—A circumscribed hypertrophy of the skin and subcutaneous tissues resulting in a softened and pendulous condition of the integument.

SYMPTOMS.—The part affected is thickened and pigmented; it is soft and fat-like to the touch; and when the condition is marked, the skin hangs in folds. The regions generally affected are the shoulders, arms, back, and buttocks.

TREATMENT.—The redundant tissue may be removed by excision or electrolysis.

KELOID.

(Cheloid, Kelis.)

DEFINITION.—A new growth resulting from hypertrophy of the connective tissue of the corium.

ETIOLOGY.—It generally results from local injury, though it is claimed that it may arise spontaneously. Certain families and individuals are especially predisposed. It is more frequent in the colored race.

SYMPTOMS.—It begins as a pale-red nodule, which slowly increases in size and sends out claw-like processes. From its resemblance to a crab it has been termed keloid. It is firm, elastic, slightly elevated, sharply defined, and ranges in size from a small bean to a growth as large as the hand. It sometimes excites pain and itching, but generally subjective phenomena are absent. The regions most frequently involved are the chest and back.

DIAGNOSIS.—Keloid may be distinguished from a *hypertrophied scar* by the fact that the latter does not extend beyond the limits of the injury.

PROGNOSIS.—The growth is usually permanent, and after removal invariably returns.

TREATMENT.—It may be removed temporarily by excision, electrolysis, or caustic pastes.

FIBROMA.

(Molluscum Fibrosum.)

DEFINITION.—A circumscribed overgrowth derived from the subcutaneous connective tissue.

ETIOLOGY—Early life and heredity are predisposing factors.

SYMPTOMS—The tumors are circumscribed; painless; soft or firm; often multiple; range in size from a pea to a hen's egg; and do not impair the general health. The overlying skin may be normal in appearance or slightly hyperæmic.

PROGNOSIS.—They are permanent and treatment is rarely indicated.

ANGIOMA.

(Nævus Vasculosus.)

DEFINITION.—A new growth, composed of cavernous tissue, or a congeries of small bloodvessels.

Angioma Cavernosum.—This form is congenital, is composed of cavernous tissue, and appears as a circumscribed, elevated, dark-red tumor, which ranges in size from a pea to one as large as the hand. It is often lobulated and pulsating.

Angioma Simplex (*Capillary Nævus, Port-wine Mark*).—This form is also congenital, and is composed of a congeries of capillaries. It is non-elevated, bright-red or purple-red in color, and may cover an area of several inches. It is generally found on the face, and constitutes what is popularly termed a *mother's mark*.

Telangiectasis.—This form is acquired, and is composed of dilated or newly-developed capillaries. It appears as a bright-red dot from which branch dilated capillaries. It is frequently associated with acne rosacæ; it is also common in those of a gouty diathesis and in those much exposed to the weather.

TREATMENT.—Cavernous angiomata may be removed by ligation, excision, or electrolysis. Simple angiomata and telangiectasis are best treated by electrolysis.

XANTHOMA.

(Vitiligoidea, Xanthelasma.)

DEFINITION.—A circumscribed connective-tissue new-growth appearing as flat patches or tubercles of a yellowish color.

ETIOLOGY.—Middle life and female sex are general predisposing factors. Hepatic disorders, especially obstructive jaundice, seem to exert a decided predisposing influence.

SYMPTOMS.—There are two forms: *Xanthoma planum*, which generally appears about the eyelids and consists of smooth, circumscribed, slightly elevated, buff-colored patches; and *Xanthoma tuberosum*, which may appear on the neck,

shoulders, trunk, or extremities, and consists of small, elastic, and yellowish-colored nodules.

TREATMENT.—These growths may be removed by excision, electrolysis, or caustics.

LUPUS ERYTHEMATOSUS.

(Seborrhœa Congestiva.)

DEFINITION—Lupus erythematosus is a new-growth resulting from a cellular infiltration of the skin, and characterized by circumscribed, red patches which are more or less covered with yellowish-gray adherent scales.

ETIOLOGY.—Middle life and female sex are predisposing factors. It frequently arises from disorders of the sebaceous glands, as seborrhœa or acne.

PATHOLOGY.—By many it is regarded as a chronic dermatitis which originates in the sebaceous glands.

SYMPTOMS.—The disease usually manifests itself on the face, in the region of the nose, and appears as small, red, slightly elevated papules, which are more or less scaly. An erythematous patch is gradually formed by the coalescence of these papules. The periphery of the patch is elevated and sharply defined, while the centre is depressed and atrophied. The ducts of the sebaceous glands are dilated and often filled with sebum. The disease spreads very slowly, shows no tendency to ulceration, and rarely excites any subjective symptoms.

DIAGNOSIS.—The location, the sharply-defined red patch with an elevated margin and depressed centre, the slight scaliness, the dilated sebaceous ducts, the chronic course, and the absence of ulceration are the diagnostic features.

Lupus Vulgaris.—This affection begins earlier in life, is characterized by tubercles and ulceration, and lacks involvement of the sebaceous glands.

PROGNOSIS.—Favorable under prolonged and judicious treatment.

TREATMENT.—General tonics like iron, arsenic, phosphorus, and cod-liver oil are often indicated.

LOCAL TREATMENT.—In many cases mild applications accomplish the most good. Much benefit is often derived

from washing the part thoroughly with green-soap and alcohol for a few days and then applying the following lotion :—

℞ Zinc. sulphatis,
Potassi sulphidi, āā ʒij ;
Aquæ, fʒiij ;
Alcoholis, fʒj.—M. (Duhring.)

Sig.—Shake well, dab the parts for fifteen minutes twice daily, and allow to dry on.

In sluggish cases stimulating applications are useful, and one of the following may be selected :—

℞ Acid. salicyl., ʒss ;
Acid. lactic., ʒss ;
Resorcin., gr. xlv ;
Zinc. oxid., ʒij ;
Vaselin. pur., ʒxvij.—M. (Broca.)

Or—

℞ Acidi pyrogallici, ʒj ;
Cerati, ʒix.—M. (Kaposi.)

Sig.—Apply locally.

In obstinate cases, scarification, curetting, or burning with the galvano-cautery may be employed with advantage.

LUPUS VULGARIS.

(Lupus Exedens.)

Definition.—A local manifestation of tuberculosis, characterized by soft red tubercles, which usually terminate in ulceration and scarring.

Etiology.—Early life and female sex are general predisposing factors. It is comparatively rare in this country, but very common in Austria and Germany. The exciting cause is the tubercle bacillus.

Symptoms.—Lupus vulgaris most frequently manifests itself on the face, especially near the nose. It begins as minute, deeply-seated, reddish-brown papules, which grow very slowly until they reach the dimensions of tubercles. They are smooth, quite soft, and seldom painful. At this stage they may either undergo slow absorption or, which is more frequent, break down and leave chronic ulcers. The ulcers are shallow, and their

edges are soft and red. There is very little discharge. They spread slowly, and may involve all the soft parts, but the bone is never invaded. While one part of the ulcer is spreading, other parts are being filled with shrivelled cicatricial tissue which in turn is often the seat of new tuberculous nodules.

DIAGNOSIS. *Epithelioma.*—Epithelioma is a disease of advanced life; it begins as a firm, wax-like nodule; the resulting ulcer starts from a single point; its borders are distinctly elevated and hard; it secretes a blood-streaked fluid; and it is often painful.

Syphilis—The age, history, associated evidences of syphilis, the rapid course, the deep ulcers, the abundant offensive discharge, and later the involvement of the bones, are the diagnostic features.

PROGNOSIS.—Very guarded. Its removal is often followed by relapse.

TREATMENT.—General tonics like iron, arsenic, phosphorus, and cod-liver oil are usually indicated.

Local Treatment.—The growth may be removed by cauterization, curetting, excision, or electrolysis. One of the following caustic applications may be employed:—

℞ Acid. arsenosi, ℈j;
Hydrarg. sulphuret. rub., ʒj;
Ung. simplicis, ʒj.—M. (HEBRA.)

Sig.—Spread thick on cloth, and apply to the patch for two or three days, until lupus nodules and points are blackish or destroyed.

Or—

℞ Acid. lactic. puri, fʒ.—M. (WICHMANN.)

Sig.—Soak a pledget of absorbent cotton and apply to the ulcer. Cover with oil-silk and bandage. Protect normal tissue with grease.

Or—

℞ Acid. salycilic., ʒij;
Adipis benzoat., ʒj.—M.

Sig.—Apply locally.

Often the best results are obtained by curetting and subsequently applying caustics.

Koch's tuberculin has lately been employed extensively in the treatment of lupus, but it has not given such good results as were expected. After its use most cases improve, many

relapse, a few recover. It seems best adapted to rapidly-spreading forms of lupus.

SYPHILIS CUTANEA.

The *secondary symptoms* appear between the first and fourth month following the chancre, and are characterized by a symmetrical arrangement, a coppery color, polymorphism (many forms at the same time), and an absence of itching. They are usually associated with certain general symptoms, such as sore throat, pain in the bones, loss of hair, enlargement of the lymphatic glands, and failure of health.

The *tertiary symptoms* appear in from six months to several years after the primary sore. They are as a rule localized, are tubercular, gummatous, or ulcerative in form, and tend to group.

Macular Syphiloderm.—This is a secondary manifestation, and consists in a general eruption of dark-red macules, varying in size from a millet-seed to a ten-cent piece.

DIAGNOSIS. *Measles.*—The absence of fever, of catarrh, of a crescentic arrangement, together with the history, will prevent an error in diagnosis.

Papular Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of large or small, dull-red papules. A few pustules are also frequently present. It pursues a chronic course, finally disappearing by desquamation, and leaving behind slight pigmentation.

DIAGNOSIS.—The history, distribution, dark color, and the presence of pustules will separate it from keratosis pilaris, papular eczema, and lichen ruber.

Tuberculous Syphiloderm.—A late manifestation, characterized by a localized eruption of dark-red shiny papules varying in size from a pea to a large bean. By some these tubercles are regarded as gummatous in character. They pursue a chronic course and finally disappear by absorption or ulceration. The ulcers thus formed, when single, are round, punched out, and frequently covered with crusts; when they coalesce, they form a serpiginous sore which pours forth a thick yellowish discharge.

DIAGNOSIS. *Lupus Vulgaris.*—This occurs in earlier life; it pursues an extremely chronic course; the ulcer is superficial; the tubercles are soft, and frequently redevelop in the scar tissue; the secretion is scant; and the bone is never involved.

Epithelioma.—In this affection the progress is slower; there is only one point of ulceration; the secretion is scanty; and the border is markedly infiltrated.

Bullous Syphiloderm.—This is a late manifestation, and is characterized by an eruption of well-filled blebs varying in size from a coffee-bean to a walnut. The contents of the blebs are puriform. They subsequently form dark, conical, stratified crusts under which are ulcers pouring forth a thick, purulent fluid.

DIAGNOSIS. *Pemphigus.*—The history, the concomitant symptoms of syphilis, and thick, greenish crusts will serve to distinguish syphilis from pemphigus.

Gummatous Syphiloderm.—This appears as a firm, circumscribed nodule which gradually turns red and softens. It may disappear by absorption, or break down and leave a deep punched-out ulcer.

Moist Papules (*Mucous Patches*).—These consist in soft flat papules covered with an offensive, grayish secretion. Heat and moisture favor their development, so that their favorite seats are around the arms, the genitalia, the mouth, and in women under the mammæ.

Papulo-squamous Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of small papules which are more or less scaly, so as to resemble psoriasis.

DIAGNOSIS.—The history, the slight scaling, the dirty-gray color of the scales, the dark-red color of the lesions, the especial tendency to involve the palms and soles will serve to distinguish syphilis from psoriasis.

Squamous Eczema.—In this affection the distribution, the infiltration of the skin, and the marked itching will lead to a correct diagnosis.

Annular Syphiloderm.—In this form the lesions consist of circles or semi-circles of small dark-red papules.

Pustular Syphiloderm.—This form usually appears within the first year, and is characterized by a general eruption of small

or large, acuminated or flat pustules which finally dry up and form yellowish-brown crusts. Large lesions leave superficial ulcers. The term *rupia* is applied to large, conical, stratified crusts which rest loosely on the ulcerating basis.

DIAGNOSIS. *Variola.*—Absence of syphilitic history, the shot-like feel, the umbilication, the itching, the high fever, and the acute course will separate variola from syphilis.

Acne.—This is usually limited to the face and shoulders; there is no history of syphilis or concomitant symptoms of that affection.

TREATMENT.—The internal treatment consists in the administration of iodide of potassium, mercurials, and tonics.

℞ Hydrarg. iodid., gr. j;
Potass. iodid., ʒiv;
Syr. sarsaparillæ co.,
Aquæ, āā fʒij.—M. (R. W. TAYLOR.)

Sig.—Teaspoonful three times a day after meals.

Or—

℞ Hydrarg. protiodidi, gr. v–x;
Ext. opii, gr. v.—M. (HARDAWAY.

Ft. in pil. No. xx.
Sig.—One morning and evening.

LOCAL TREATMENT.—Papular eruptions may be washed with mercurial lotions; mucous patches may be dusted with calomel; ulcers may be dressed with iodoform.

LEPROSY.

(Lepra, Elephantiasis Græcorum.)

DEFINITION.—A chronic contagious disease, excited by the bacillus of leprosy, and characterized by tubercular formations, ulcerations, atrophy, disturbances of sensation, and an increase or decrease of pigment.

ETIOLOGY.—The disease is contagious, but direct inoculation is essential to its transmission. It seems to be more common in hot climates. The exciting cause is the bacillus lepræ, which closely resembles the tubercle bacillus.

VARIETIES.—There are two varieties: Tubercular leprosy and anæsthetic leprosy; but the two forms are often associated in the same patient.

Symptoms.—Certain prodromes may precede the outbreak of the disease, such as malaise, headache, chilliness, depression of spirits, and numbness in the parts to be affected.

Tuberculur Leprosy.—In this form spots of erythema appear on the body; they soon become pigmented and hyperæsthetic, and develop into tubercles varying in size from a pea to a walnut. The face, extremities, and genitals are the parts most commonly affected, but occasionally the mucous membranes, especially of the nose and throat, are invaded. Ultimately the tubercles may break down and leave superficial indolent ulcers. In some cases a bullous eruption appears from time to time. The hair, eyebrows, and eyelashes fall out, the eyes become inflamed, the features distorted, and the voice husky. The disease may last many years, death finally resulting from exhaustion or some intercurrent disease.

Anæsthetic Leprosy.—In this form the peripheral nerves are invaded by the bacillus lepræ. The outbreak may be preceded by numbness, itching, or lancinating pains. These symptoms are followed by the appearance of discolored spots, which are at first associated with hyperæsthesia, but later more or less anæsthesia develops. The skin and its appendages atrophy, the bones undergo necrosis, and the phalanges drop off one by one. In some cases (lepra alba) the skin is not only anæsthetic, but distinctly white. Finally, when the nerves are more or less destroyed paralysis results. The duration is many years.

Prognosis.—Unfavorable. A cure is practically impossible, though the progress of the disease may be stayed by appropriate treatment.

Treatment.—Sufferers should be isolated. Tonics are usually indicated. Chaulmoogra oil and gurgun oil, internally and externally, have been highly recommended. Externally, chrysarobin, ichthyol, or resorcin may be applied to the affected parts.

℞ Chrysarobin, gr. x–ʒj;
Ætheris et alcoholis ad q. s.
Collodii, fʒj.—M. (G. H. Fox.)

Rub the chrysarobin with a little alcohol and ether, and add the collodion.

Sig.—Paint the affected patch with a camel's-hair brush.

EPITHELIOMA.

(Skin Cancer.)

Etiology.—Late life, heredity, and local irritation are the predisposing factors.

Varieties.—Superficial, deep-seated, and papillomatous.

Superficial Epithelioma (*Rodent Ulcer*).—This form usually begins as a firm, circumscribed, reddish-yellow, wax-like papule. After the lapse of several months or years the papule becomes scaly, and the removal of the scales is followed by a slight excoriation, which in turn becomes covered with a slight, reddish-brown crust. The latter tends to adhere, and its repeated removal is followed by a raw surface, which is gradually converted into an ulcer. The ulcer has a prominent indurated margin; its outline is irregular; its base is uneven and glazed; and it exudes a sanious viscid excretion. It is not painful; it does not lead to enlargement of the neighboring lymphatic glands; nor does it cause impairment of the general health. It spreads very slowly, and sometimes becomes stationary or actually heals. More frequently the ulceration continues until it involves all the tissues of the part, even the bones. The ulcer generally appears on the face, and in its advance it may destroy the nose, eyes, or a large portion of the cranial bones.

Deep-seated Epithelioma.—This variety may begin as a deep-seated, red, shiny tubercle, or it may develop from the superficial form. The ulcer which is ultimately formed is deep; its base is granular; its edges are everted, indurated, and of a reddish-purple color; it secretes a blood-stained yellow fluid; it is the seat of lancinating pain; it causes enlargement of the neighboring glands; and it sooner or later induces the cancerous cachexia. Death may result from exhaustion, or more rarely, from hemorrhage caused by ulceration of a large bloodvessel.

Papillomatous Epithelioma.—This may begin as a warty excrescence, or may develop from one of the preceding varieties. It is characterized by an ulcerated surface from which springs an aggregation of large, highly-vascular papillæ. Be-

tween the papillæ there are often deep-seated fissures from which exudes an offensive viscid discharge. The general health is impaired and the neighboring glands are enlarged.

Diagnosis. *Lupus Vulgaris.*—Lupus begins in the young; the original papule is soft; there is often more than one centre of ulceration; the margins of the ulcer are not hard and everted; the progress is extremely slow; the discharge from the ulcer is very scant, and the bones are never involved.

Syphilis.—The history, the associated evidences of syphilis, the rapid progress of the ulceration, the abundant discharge, the absence of pain, and the effect of treatment will suggest the diagnosis.

Prognosis.—Guarded. A thorough removal in the beginning of the disease is often followed by a permanent cure. When the process is advanced the growth usually returns.

Treatment.—Epitheliomatous growths may be removed by the use of caustics, the cautery, the curette, or by excision. The last is preferable when the growth is small and circumscribed.

AINHUM.

Ainhum is a rare affection, occurring chiefly in the colored race, and characterized by the appearance of a groove or furrow at the base of one or more of the toes. The groove deepens, the affected member becomes swollen, and finally drops off at the point of strangulation.

DERMATALGIA.

Dermatalgia, or neuralgia of the skin, is a rare affection, and is characterized by paroxysms of sharp, lancinating pain in the skin, which arise without any change in the local appearance. It is most frequently observed in women of a neuropathic tendency, and may arise from any of the causes which induce neuralgia elsewhere.

Treatment.—The cause must be sought for and, if possible, removed. Tonics like iron, arsenic, quinine, and phosphorus are often indicated. Locally, massage and electricity may prove useful.

PRURITUS.

DEFINITION.—Pruritus is a functional affection, characterized by itching which is unassociated with any objective phenomena.

ETIOLOGY—Pruritus may arise without obvious cause, as the *Pruritus senilis* observed in the old, and the *pruritus hiemalis* which develops on the approach of cold weather and disappears when the weather becomes warm.

Symptomatic Pruritus.—Pruritus may be a symptom of many conditions, notably diabetes, gout, lithæmia, hysteria, neurasthenia, and Bright's disease.

SYMPTOMS.—There is only one symptom and that is itching; but as a result of scratching, the part may become hyperæmic, thickened, or the seat of eczema.

DIAGNOSIS.—Pruritus must be distinguished from the itching induced by *pediculosis, or some local disease, like eczema.*

PROGNOSIS.—This will depend on the cause. When the primary disease is curable the prognosis for permanent relief is favorable. In other cases temporary relief only is to be expected.

TREATMENT.—Search should be made for the exciting cause, which should be removed, if possible. In all cases the urine must be examined for sugar, since diabetes is one of the most frequent causes of pruritus. Among the internal remedies recommended for pruritus may be mentioned nux vomica, belladonna, and pilocarpine. The best local remedies are carbolic acid, vinegar, thymol, chloral-camphor, boric acid, hydrocyanic acid, hot water, and menthol.

℞ Acid. hydrocyan. dil., f℥ij;
Sodii borat., ℥j;
Aq. rosæ, f℥viij.—M. (FOX.)
Sig.—Use locally.

℞ Menthol, ℥iss;
Alcoholis, f℥iv.—M.
Sig.—Use locally.

℞ Acid. carbolic., f℥j–f℥ij;
Aquæ et alcohol., āā q. s. ad Oj.—M.
Sig.—Apply locally as often as necessary.

TINEA TRICOPHYTINA.

(Ringworm.)

DEFINITION.—A contagious disease excited by a vegetable parasite—the tricophyton.

VARIETIES.—On the scalp it is termed *Tinea tonsurans;* on the body, *Tinea circinata;* on the bearded region, *Tinea sycosis.*

Tinea Tonsurans.

This form is observed almost exclusively on the scalp of children. It is characterized by one or more rounded, scaly, elevated, grayish-colored patches through which project dry, brittle, lustreless, broken-off hairs.

DIAGNOSIS. *Seborrhœa.*— The patches are not circumscribed; the scales are greasy; the hair is not involved; and the microscope reveals no parasite.

Eczema.—The patches are not circumscribed; the hair is not involved; there is more inflammation; there is marked itching; and the microscope reveals no parasite.

Alopecia Areata.—Baldness is complete; there are no scales; and the base is smooth and shiny.

PROGNOSIS.—Favorable.

TREATMENT. — Tonics are often indicated. The parts should be thoroughly washed with soap and water, and the affected hairs removed. The following parasiticides may be employed in ointment or lotion; mercury, sulphur, chrysarobin, or sulphurous acid.

℞ Acid. sulphurosi, f℥j;
Aquæ, f℥iv.—M.
Sig.—Apply several four or five times daily.

Or—

℞ Beta naphthol, gr. xl.;
Sulphuris præcip., ℥j;
Vaselini, ℥j.—M.
Sig.—Rub into affected area once or twice daily. (Hardaway.)

Tinea Circinata.

(Ringworm of the Body.)

This appears as one or more rounded, red, slightly-elevated scaly patches, which on close examination reveal minute vesicles or papules. As the disease advances new patches spring from the periphery while the central portion clears up. There is often considerable itching.

DIAGNOSIS. *Psoriasis.*—The marked scaling; the absence of itching; the tendency to involve the extensor surfaces, especially the knees and elbows; and the absence of the tricophyton will separate psoriasis from ringworm.

Eczema.—The patches are ill defined; do not clear in the centre; there is more infiltration of the skin; and there is no tricophyton.

PROGNOSIS.—Favorable.

TREATMENT.—Tonics are frequently indicated; mercury, sulphur, sulphurous acid, and hyposulphite of sodium are among the best parasiticides.

℞ Sodii hyposulphit., ʒij;
Aquæ, fʒij.—M. (DUHRING.)

Sig.—Apply locally.

Or—

℞ Hydrarg. ammoniat., gr. xxx;
Adipis, ʒj.—M.

Sig.—Apply locally.

Tinea Sycosis.

(Barber's Itch, Sycosis Parasitica.)

This begins as a red scaly patch involving the bearded region. Soon purplish tubercles and pustules form around the opening of the hair-follicles, and the hairs become lustreless, brittle, and loose. There is often considerable itching.

DIAGNOSIS. *Simple Sycosis.*—In this the inflammation is superficial; the hairs are not involved; and the tricophyton is absent.

Eczema.—The tubercles, the involvement of the hairs, and the presence of the tricophyton will separate it from eczema.

Prognosis.—Favorable; unless treated actively, however, there may be a permanent loss of hair.

Treatment.—The affected hairs should be removed, and one of the following parasiticides employed in lotion or ointment: Mercury, sulphur, or hyposulphite of sodium.

℞ Sodii hyposulphit., ʒiij;
Aquæ, fʒiij.—M.
Sig.—Apply locally.
Or—

℞ Sulphur. sublimat., ʒij;
Vaselini, ʒij.
Sig.—Apply locally.

TINEA VERSICOLOR.

(Pityriasis Versicolor.)

Definition.—A chronic affection excited by a vegetable parasite, the *microsporon furfur*, and characterized by fawn-colored scaly patches which usually appear about the chest.

Etiology.—It is a disease of adult life, and is more frequently observed in the debilitated and uncleanly.

Symptoms.—It appears usually on the front of the chest as small round spots of a pale-yellow or fawn color, which slowly enlarge, fuse, and form slightly-elevated scaly patches. Subjective symptoms are generally absent.

Diagnosis.—*Chloasma* somewhat resembles tinea versicolor; but the former is not often observed on the trunk, is not scaly, and is not associated with a parasite.

Prognosis.—Favorable.

Treatment.—The parts should be frequently washed with soap and water, after which one of the following parasiticides may be applied: Corrosive sublimate (gr. ij to an ounce of water), sulphurous acid, or hyposulphite of sodium:—

℞ Sodii hyposulphitis, ʒv;
Glycerini, fʒiij;
Aquæ, q. s. ad fʒv.—M.
Sig.—Apply locally.

Or—

℞ Hydrarg. chlor. corros., ℈j;
Alcoholis, f℥iv;
Saponis viridis, ℥ij;
Ol. lavandulæ, f℥j.—M. (Van Harlingen.)

Sig.—To be rubbed in well night and morning.

TINEA FAVOSA.

(Favus.)

Definition.—A contagious affection of the scalp excited by the *achorion Schönleinii*, and characterized by yellowish, cup-shaped crusts.

Etiology.—It is observed especially in poor, ill-nourished children.

Symptoms.—The disease is characterized by one or more rounded, yellow, cup-shaped crusts, through which project dry, brittle, lustreless hairs. The underlying tissue is more or less atrophied and scarred. It is associated with some itching and a peculiar musty odor.

Diagnosis.—The yellow, cup-shaped crusts, the odor, and the atrophy of the skin will separate it from *ringworm*.

Prognosis.—Favorable When not treated early it may be followed by permanent baldness.

Treatment.—The crusts should be removed by oil, or soap and water. The affected hairs should also be removed. The following parasiticides are efficient: Mercury, sulphur, chrysarobin, and hyposulphite of sodium.

SCABIES.

(Itch.)

Definition.—Scabies is a contagious disease excited by an animal parasite—the *Acarus Scabiei*—and manifested by papules, vesicles, pustules, burrows, and intense itching.

Etiology.—The disease is always acquired through intimate intercourse with patients already affected.

Symptoms.—The disease manifests itself by intense itching, which is associated with an eruption of small papules, vesicles,

and pustules. Among these lesions may be found cuniculi, or burrows; these are discolored, dotted, slightly elevated lines ranging from a line to half an inch in length, and produced by the penetration of the female acarus and the deposition of her eggs along the passage. The parts most commonly affected are the hands between the fingers, the wrists, the axillæ, the genitalia, beneath the mammæ, and the inner aspects of the thighs. The face and scalp are never involved.

DIAGNOSIS.—The recognition of scabies rests on the history, the itching, the presence of burrows, the multiformity of the lesions, and their peculiar distribution.

PROGNOSIS.—Favorable.

TREATMENT.—Ointments of sulphur, styrax, and naphthol are efficient remedies. After a thorough bath the whole body should be anointed twice daily for three or four days. At the end of this time the bath should be repeated, and the bed linen and underclothing changed. The infected clothing should be sterilized.

℞ Sulphur. sublimat., ʒj;
Balsam. Peruvian., ʒss;
Adipis, ʒj.—M. (DUHRING.)
Sig.—Rub in thoroughly twice daily.

℞ Styracis liquid., ʒiv;
Adipis, ʒiss.—M.

PEDICULOSIS.

(Phtheiriasis.)

Pediculosis Capitis.—This form results from the pediculus capitis, or head-louse, a gray insect from one to two millimetres in length. The condition is recognized by itching of the scalp and the discovery of the lice or their white ova, or nits. Eczematous lesions resulting from scratching are often observed.

Pediculosis Corporis.—This form results from the pediculus corporis, pediculus vestimenti, or body-louse, a somewhat larger insect than the head-louse. The condition is recog-

nized by intense itching on the covered parts of the body, scratch-marks, petechiæ caused by the bite of the insect, and the discovery of the lice on the garments.

Pediculosis Pubis.—This form results from the pediculus pubis, or crab-louse, a minute, gray, translucent insect. It is found on parts covered with short hair, as the pubes, axillæ, eyebrows, etc.

TREATMENT.—In *pediculosis capitis* the head may be thoroughly washed with coal-oil, dilute carbolic acid (℥j to Oj), or tincture of cocculus indicus.

In *pediculosis corporis* the parts should be thoroughly washed and the clothes subjected to a high temperature. The body may be bathed in a weak solution of corrosive sublimate.

In *pediculosis pubis* an ointment of mercury is very efficient.

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